The Journal of
Thoracic Surgery

VOL. 30 OCTOBER, 1955 NO. 4

Original Communications

THE USE OF PRESSURE RECORDINGS OBTAINED AT TRANSTHORACIC LEFT HEART CATHETERIZATION IN THE DIAGNOSIS OF VALVULAR HEART DISEASE

DON L. FISHER, M.D.* (BY INVITATION)
PITTSBURGH, PA.

INTRODUCTION

SINCE the introduction of a method of transthoracic left atrium puncture by Viking Olov Björk,[1] the procedure has been further adapted to include the insertion of a plastic catheter for access to the left ventricle and aorta.[2, 3] Once this access has been gained, studies of cardiac output, dye injection, intracardiac electrocardiography, angiocardiography, and pressure recording can obviously be applied, as in right heart catheterization techniques. However, the principal use has been in the recording of pressures for the demonstration of the effects of valve lesions. Such pressure recordings may supply a need that sometimes cannot reliably be met by the many other diagnostic methods which are available.

This report of an initial series of 61 cases illustrates the use of left heart catheterization as a preoperative diagnostic test. Nearly all the patients had rheumatic valve disease, with uncertain or atypical combinations of findings on auscultation, electrocardiography, and fluoroscopy. Diagnostic criteria for judging the pressure effects of specific valve lesions were derived from recordings of pressure in experimental lesions presented in the literature, from previous experience with direct recordings at thoracotomy,[4] and from recordings of the analogous tricuspid and pulmonic valve lesions observed at right heart catheterization (Table I.).

METHODS

The 61 tests were applied to 6 outpatients and 52 inpatients. Premedication was given in the fasting state. Moderate sedation in the average adult was produced by a dosage of pentobarbital, 0.1 Gm., orally, and meperidine

Read at the Thirty-fifth Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, N. J., April 24 to 26, 1955.

*Director, Cardiopulmonary Laboratory, Allegheny General Hospital, Pittsburgh, Pa.

TABLE I. TENTATIVE CRITERIA FOR DIAGNOSIS BY PRESSURE EFFECTS

	SEVERITY				
	0	1+	2+	3+	4+
Mitral Stenosis Effect.— Left atrium mean pressure minus left ventricle early diastolic pressure (early diastolic pressure immediately follows the initial diastolic dip) (mm. Hg)	0-3	4-6	6-10	10-15	15+
Mitral Insufficiency (or Incompetence) Effect.— Left atrium V-wave height, minus C-wave height	0-3	4-6	6-8	8-10	10+
Aortic Stenosis Effect.— Left ventricle systolic pressure, minus aorta or peripheral artery systolic pressure	±15	15-20	20-25	25-30	30+
Aortic Insufficiency.—Not studied					
Left Ventricular Failure Effect.— Left ventricle end-diastolic pressure minus upper limit of normal (9 mm.)	0	1-3	4-6	7-9	9+

hydrochloride, 100 mg., intramuscularly. Limb electrodes were connected for continuous monitoring of the electrocardiogram on the cathode-ray oscilloscope. An indwelling needle or percutaneous arterial catheter was placed in a peripheral artery, the lumen being kept free by intermittent flushing or with a blunt stylus. In 6 patients, a right heart catheter was also placed, through an antecubital vein, for "pulmonary capillary" pressure determinations simultaneously with the direct left atrium pressures. The patient was then turned to a prone position on the padded fluoroscopy table, and the skin of the back was scrubbed and draped. The underside of the fluoroscope screen was draped with a sterile towel. At a brief preliminary fluoroscopy, the appropriate skin site for needle puncture was chosen, directly opposite the position of the left atrium, using a hemostat for a pointer. This was usually in the right seventh or eighth intercostal space, at a point 2 fingerbreadths (4 to 4½ cm.) to the right of the spinous processes. The skin and subcutaneous tissues were infiltrated with 4 c.c. of 1 per cent procaine, and then a 6-inch No. 18T thin-walled styletted needle was inserted. It was directed anteriorly, and inclined about 25 degrees obliquely to the left, so as to pass in front of the spine by a right paravertebral approach. Contact with bone was avoided, in order to prevent periosteal pain.

The position of the needle was checked fluoroscopically at a needle depth of 7 to 8 cm., and it was reinserted if necessary. The left atrium was entered at a needle length of 10 to 14 cm. Bright red blood then issued, or could be aspirated easily. A blood sample was withdrawn for blood gas measurements. If dark blood was aspirated, then right heart catheterization was first performed (three cases). The left atrium was then located, during either the withdrawal or reinsertion of the needle.

Recordings of pressure were made directly from the needle, showing the contour of the atrial pressure curve at high sensitivity, using the Hathaway PS-8AI inductance gauge, with Sanborn strain amplifier and direct writing recorder. The needle was repeatedly flushed with heparinized 0.9 per cent

sodium chloride solution to prevent thrombus formation. A plastic catheter* was next inserted, to gain access to the left ventricle and aorta. The pressure was monitored during this maneuver, and no attempt was made to visualize the catheter fluoroscopically. Several recordings were made of the pressure transition during withdrawal of the catheter through the aortic and mitral valves. If the aorta could not be entered, then recordings were made from the left ventricular position simultaneously with the peripheral artery recording from needle or catheter. The intracardiac catheter and left atrium needle were then withdrawn, having been left in place 3 to 75 minutes (average 30 minutes). The pressure gauges were calibrated against mercury and water manometers. Corrections were applied to the various pressure measurements so as to correspond with a zero reference level at the intra-atrial point of the needle. Physical examination and chest films were repeated at twenty-four hours to detect possible complications.

RESULTS

A comparison of the interpretations of pressure measurements with findings at operations and autopsies is recorded in Table II. There was only one major disagreement in these respective diagnoses, using the criteria for the preoperative diagnosis as listed in the table.

In the 6 patients who also had simultaneous right heart catheterization by the venous route, the pulmonary artery wedge pressure corresponded closely to the left atrial pressure in both pulse contour and mean pressure level in 3 cases. It corresponded only with the mean pressure level and not in pulse contour, in 3 cases.

In 2 cases there was a definite anacrotic (upstroke) notching and delay on the brachial artery pressure recording, yet no systolic pressure drop was present across the aortic valve at left heart catheterization, and no systolic aortic thrill was palpable at operation. However, in each case of aortic stenosis with systolic pressure drop across the valve, there was some deformity of the brachial artery systolic peak.

Complications occurred in Patients 1, 7, and 14 of the series. In Patients 1 and 7, the needle was first directed into the right hilum, entering the pulmonary artery. Minor fluid densities were seen later at the right bases on x-ray films. At a thoracentesis on Patient 7, 125 c.c. of blood was aspirated. The asymptomatic hemothorax cleared spontaneously thereafter, in each of these 2 cases. In Patient 14, the needle was directed too far cephalad, entering the base of the aorta. Mild signs of acute pericardial tamponade occurred, followed by signs and symptoms of chronic tamponade during the next six weeks. Eight pericardiocenteses were done, beginning on the second post-test day. No hematologic abnormalities were found except a positive tourniquet test (Rumpel-Leede) and slight bleeding of gingivae; but the hemopericardium regressed coincidentally with vitamin C—vitamin P treatment (CVP capsules, twice daily). Patient 45 required ¼ grain morphine, subcutaneously, for

*Polyvinyl tubing No. 442T. Becton-Dickinson and Company, Rutherford, N. J.; or special polyethylene tubing, Irvington Varnish and Insulator Co., Irvington, N. J.

TABLE II. COMPARISON OF DIAGNOSIS OF VALVE LESIONS BY VARIOUS MEANS

TEST NO.	PT.	AGE	RHYTHM	RATE	PRESSURES, MM. HG				PRESSURE EFFECTS			CLINICAL DIAGNOSIS			SURGICAL DIAGNOSIS			AUTOPSY DIAGNOSIS		
					L.A.	L.V.	AORTA	PERIPHERAL ARTERY	M.S.	M.I.	A.S.	M.S.	M.I.	A.S.	M.S.	M.I.	A.S.	M.S.	M.I.	A.S.
1	C. B.	30	A.F.	135	35	–	–	–	–	6½ mm.	–									
									–	2+	–	2+	3+	0	–	–	–	–	–	–
2	P. F.	33	N.	96	22	–	–	–	–	0	–	3+	2+	0	4+	0	0	4+	0	0
3	V. P.	44	A.F.	75	36	–	–	–	–	7½	–									
									–	2+	–	3+	1+	0	–	–	–	–	–	–
4	A. E.	46	A.F.	90	16	–	–	–	–	3	–									
									–	0	–	2+	1+	3+	–	–	–	–	–	–
5	J. M.	22	N.	92	31	–	–	–	–	10	–					(myxoma)				
									–	4+	–	3+	1+	0	?	?	0	–	–	–
6	M. C.	38	A.F.	68	15	–	–	–	–	3½	–									
									–	1+	–	2+	3+	1+	–	–	–	–	–	–
7	V. M.	36	N.	83	18	211/18	–	140/98 (cuff)	0	0	71									
									0	0	4+	2+	0	4+	0	0	4+	0	0	4+
8	A. H.	50	A.F.	83	31	–	–	–	–	13½	–									
									–	4+	–	2+	2+	2+	–	–	–	–	–	–
9	D. R.	36	N.	85	31	–	–	–	–	3	–									
									–	0	–	3+	1+	0	4+	0	0	–	–	–
10	J. S.	31	N.	85	28	100/3	–	–	25	0	–									
									4+	0	–	3+	1+	0	4+	0	0	4+	0	0
11	S. P.	15	A. F.	100	33	–	–	–	–	21	–									
									–	4+	–	–	4+	–	–	–	–	0	4+	0
12	A. G.	57	A.F.	90	29	–	–	–	–	0	–									
									–	0	–	3+	2+	0	–	–	–	–	–	–
13	H. O.	57	N.	70	23	–	–	–	–	0	–									
									–	0	–	3+	1+	0	–	–	–	–	–	–
14	P. D.	27	N.	120	–	–	122/88	–	–	–	–	0+	2+	0	–	–	–	–	–	–
15	M. B.	46	A.F.	115	25	–	–	–	–	0	–									
									–	0	–	2+	3+	3+	–	–	–	4+	0	1+
16	F. E.	57	A.F.	75	13	121/6	95/58	–	7	0	26									
									2+	0	2+	3+	0	1+	–	–	–	–	–	–
17	V. W.	39	N.	80	26	103/9-13	–	–	17	0	–									
									4+	0	–	3+	2+	0	4+	0	0	–	–	–
18	M. T.	48	A.F.	90	24	96/14	–	–	10	4	–									
									3+	1+	–	2+	0	0	–	–	–	–	–	–
19	E. C.	27	A.F.	95	26	106/9	–	107/76	17	0	0									
									4+	0	0	3+	2+	0	4+	0	0	–	–	–
20	L. S.	35	N.	60	17	124/5-11	–	–	12	0	–									
									3+	0	–	3+	1+	2+	–	–	–	–	–	–
21	H. M.	41	N.	75	25	190/15	–	96/56	10	4	94									

21	H. M.	41	N.	75	25	190/15	-	96/56	10	4	94								
									3+	1+	4+	2+	0	4+	2+	1+	4+	-	-	-
22	M. P.	24	A.F.	65	30	125/11	-	127/85	19	0	0									
									4+	0	0	2+	2+	2+	4+	0	0	-	-	-
23	M. B.	23	A.F.	80	14	100/9-13	-	93/76	5	0	7									
									1+	0	0	1+	3+	2+	-	-	-	-	-	-
24	M. Z.	46	N.	90	15	152/14	-	118/59	1	0	34									
									0	0	3+	2+	0	3+	-	-	-	-	-	-
25	D. B.	51	A.F.	50	25	-	-	117/87	-	4½	-									
									-	1+	-	2+	3+	2+	-	-	-	-	-	-
26	A. L.	55	A.F.	95	14	-	-	-	-	0	-									
									-	0	-	3+	2+	0	-	-	-	-	-	-
27	A. L.	55	A.F.	85	8	105/2-6	-	-	6	0	-									
									1+	0	-	3+	2+	0	-	-	-	-	-	-
28	D. B.	51	A.F.	50	26	-	-	-	-	6	-									
									-	2+	-	2+	3 +	2+	-	-	-	-	-	-
29	T. V.	15	N.	140	22	110/6	110/80	-	16	9	0									
									3+	3+	0	2+	3+	0	-	-	-	-	-	-
30	L. H.	59	N.	95	28	95/6-10	-	-	22	15	-									
									4+	4+	-	2+	3+	0	-	-	-	-	-	-
31	O. S.	44	A.F.	70	23	106/6-7	-	-	17	5	-									
									4+	1+	-	2+	3+	0	-	-	-	-	-	-
32	A. S.	36	A.F.	90	21	100/4-7	-	-	17	0	-									
									4+	0	-	3+	2+	0	4+	1+	0	-	-	-
33	J.L.	26	A.F.	70	22	136/8-10	98/73	-	14	3½	34									
									3+	1+	3+	3+	2+	0	-	-	-	-	-	-
34	N. H.	32	N.	70	21	115/10	-	111/56	11	0	4									
									3+	0	0	3+	1+	1+	4+	0	0	-	-	-
35	T. K.	45	N.	80	26	107/5	107/76	-	21	0	0									
									4+	0	0	1+	3+	0	3+	2+	0	-	-	-
36	R. T.	49	A.F.	75	28	165/9	-	-	18	3½	-									
									4+	1+	-	3+	2+	0	-	-	-	-	-	-
37	J. H.	36	N.	90	40	154/7	-	-	33	4	-									
									4+	1+	-	2+	3+	1+	-	-	-	-	-	-
38	E. Y.	32	N.	90	25	117/0	-	-	25	0	-									
									4+	0	-	3+	2+	0	4+	0	0	-	-	-
39	R. K.	40	A.F.	65	20	95/12-15	-	-	8	13⅓	-									
									2+	4+	-	1+	3+	2+	-	-	-	-	-	-
40	M. C.	28	N.	80	33	160/5-8	-	119/67	28	7½	41									
									4+	2+	3+	2+	2+	2+	3+	1+	3+	-	-	-

Pressure effects are expressed both in mm. Hg, and as 0 to 4+ grades of severity, as in Table I.
L.A.=left atrium.
L.V.=left ventricle.
A.F.=atrial fibrillation.
N=normal sinus rhythm.
M.S.=mitral stenosis.
M.I.=mitral insufficiency and incompetence.
A.S.=aortic stenosis.
No cases of aortic insufficiency were studied.

TABLE II—CONT'D

TEST NO.	PT.	AGE	RHYTHM	RATE	PRESSURES, MM. HG				PRESSURE EFFECTS			CLINICAL DIAGNOSIS			SURGICAL DIAGNOSIS			AUTOPSY DIAGNOSIS		
					L.A.	L.V.	AORTA	PERIPHERAL ARTERY	M.S.	M.I.	A.S.	M.S.	M.I.	A.S.	M.S.	M.I.	A.S.	M.S.	M.I.	A.S.
41	J. M.	49	A.F.	60	30	100/4-8	–	–	26	0	–									
									4+	0	–	3+	0	0	4+	0	0	4+	0	0
42	J. F.	52	N.	72	11	114/10-11	–	114/90	1	3	0	(Constr.								
									0	0	0	pericarditis)			–	–	–	–	–	–
43	E. B.	49	N.	85	8½	120/3-7	120/75	–	5½	0	0									
									1+	0	0	1+	3+	0	–	–	–	–	–	–
44	E. R.	34	A.F.	80	30	–	–	–	–	9	–									
									–	3+	–	1+	3+	0	2+	3+	0	–	–	–
45	C. H.	41	A.F.	100	26	100/2-6	–	–	24	6½	–									
									4+	2+	–	2+	3+	0	3+	2+	0	–	–	–
46	P. S.	43	N.	90	26	99/7-9	–	98/68	19	0	0									
									4+	0	0	4+	1+	0	4+	0	0	–	–	–
47	O. D.	40	N.	80	15	115/9	–	107/81	6	0	8									
									1+	0	0	3+	2+	1+	4+	0	1+	–	–	–
48	T. V.	16	N.	120	18	–	–	–	–	8	–									
									–	3+	–	2+	3+	0	–	–	–	–	–	–
49	F. G.	52	N.	75	20	100/8-10	–	–	12	0	–									
									3+	0	–	4+	0	0	4+	1+	0	–	–	–
50	R. C.	36	N.	60	13	108/2-5	–	110/64	11	0	0									
									2+	0	0	3+	0	0	–	–	–	–	–	–
51	D. T.	40	A.F.	90	13½	106/4-6	–	100/75	9½	0	6									
									2+	0	0	2+	3+	0	3+	1+	0	–	–	–
52	C. C.	36	N.	105	20	107/5-8	–	109/77	15	4	0									
									4+	1+	0	3+	2+	0	–	–	–	–	–	–
53	J. W.	37	N.	70	26	110/2-7	–	98/58	24	0	12									
									4+	0	0	3+	0	2+	4+	0	0	4+	0	0
54	R. Z.	40	A.F.	60	17	130/5-8	–	100/68	12	3½	30									
									3+	1+	3+	2+	3+	1+	3+	1+	2+	–	–	–
55	E. A.	45	A.F.	60	25	96/4-8	–	106/72	21	2	0									
									4+	0	0	3+	0	1+	–	–	–	–	–	–
56	J. B.	33	N.	70	6	111/2-6	111/71	113/75	4	0	0									
									1+	0	0	2+	0	0	–	–	–	–	–	–
57	J. B.	20	A.F.	95	39	136/0-3	–	–	39	5	–									
									4+	1+	–	2+	3+	0	4+	1+	0	–	–	–
58	A. S.	32	A.F.	120	11	121/7-9	–	135/79	4	0	0									
									1+	0	0	3+	1+	0	–	–	–	–	–	–
59	E. C.	49	A.F.	65	18	95/2-5	–	–	16	4	–									
									4+	1+	–	4+	0	0	4+	0	0	4+	0	0
60	D. J.	27	A.F.	65	12	98/10-12	–	110/74	2	0	0									
									0	0	0	1+	3+	0	–	–	–	–	–	–
61	H. D.	31	N.	70	15	90/4-8	–	95/60	11	0	0									
									3+	0	0	2+	2+	0	2+	3+	0	–	–	–

(For footnote see p. 383.)

relief of severe pleuritic chest pain after the test, although no evidence of organic cause was found on repeated auscultation, fluoroscopy, and x-ray films.

No emboli were dislodged, although there was history of embolization previously, during cardiotomy, or postoperatively, in 6 patients. No pneumothorax occurred. There was no evidence of esophageal injury.

Samples of pericardial fluid were sought for study at subsequent thoracotomy in 16 patients, and in 8 of these the fluid contained hemoglobin, 1 to 5 Gm. per cent.

The needle was unintentionally lodged in vascular structures other than the left atrium in 10 patients, as listed in Table III. No complications resulted therefrom, except as previously noted. Right heart catheterization through the needle was performed three times as a result of such location in the right atrium or vena cava, as an additional procedure.

TABLE III. ADDITIONAL VASCULAR ORGANS ENCOUNTERED AT LEFT ATRIUM PUNCTURE

PATIENT NO.	STRUCTURES
1	Superior vena cava; also pericardial cavity (clear fluid)
7	Right pulmonary artery
14	Base of aorta; also pericardial cavity (clear fluid)
16	Base of aorta
17	Right atrium
20	Right atrium
24	Intercostal artery
42	Right atrium
54	Right atrium
56	Right atrium; also pericardial cavity (clear fluid)

(For footnote see p. 383.)

DISCUSSION

Estimation of the severity of valvular lesions by standard clinical and laboratory tests may sometimes be grossly in error. Therefore, judgment of the reliability of the pressure tests must be based on comparisons with surgical diagnoses by means of intracardiac and extracardiac palpation, and with autopsy findings. In this series of cases, the comparisons show good agreement. In all cases (except one) where an operation had been performed, the diagnosis as to the relative severity of the multiple lesions and the decision as to the proper surgical approach were correctly influenced by the pressure interpretations.

There were several unimportant discrepancies in the grading of cases of minor mitral insufficiency; and in one patient (No. 61), a major lesion of mitral insufficiency was not detected. This error occurred with relatively low left atrial mean pressure. It seems likely that the specific pressure effects of mitral stenosis or insufficiency will be exaggerated when the left atrium is distended with higher pressure. A less tense atrium and pulmonary vascular bed would result in less pressure rise from a given regurgitant jet.

Although the test has been quite reliable in this series, much further experience is needed for proper evaluation of diagnostic standards. The im-

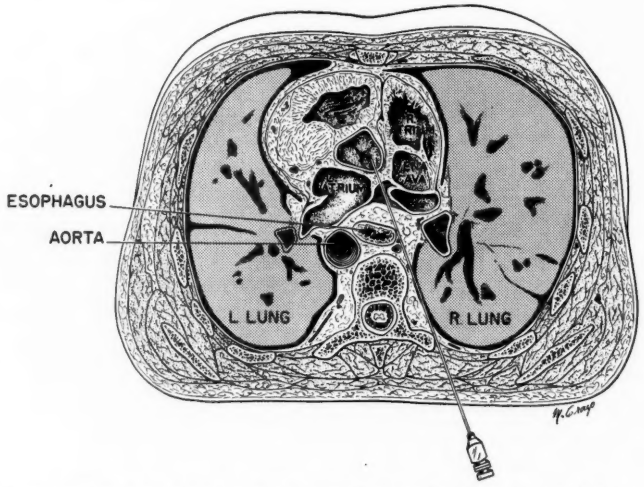


Fig. 1*A*.—Cross-section view showing atrial puncture needle directed too high, entering the base of the aorta.

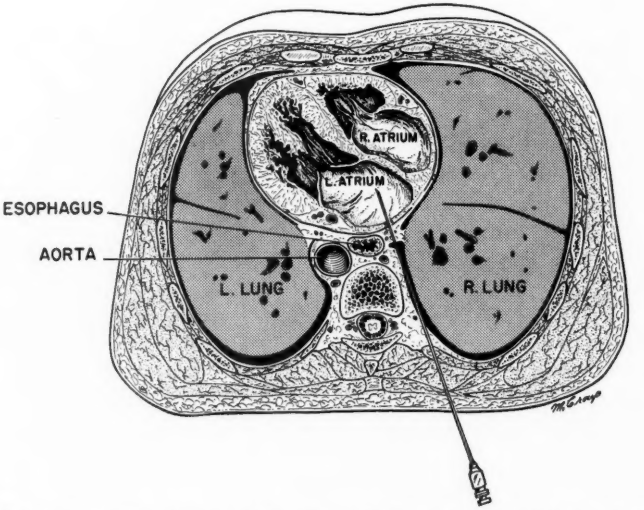


Fig. 1*B*.—Cross-section view showing the needle properly directed into the left atrium. Further advance will encounter the right atrium. (Fig. 1*B*, reprinted by permission of the Editor, *Annals of Surgery*.)

portant factors of heart rate and rhythm, pulmonary blood volume and vascular distensibility, and cardiac output should be considered in measuring pressure effects.

The procedure of transthoracic left atrium puncture can be varied in many other ways to suit individual requirements. The technique of Björk has been modified in this laboratory so as to use fluoroscopic guidance of the needle. With standard fluoroscopic equipment, this requires use of the prone position for insertion of the needle. The pressure recordings of this study were made in such position. If recordings in other reclining positions are desired, the needle alone can be removed over the flexible catheter, and then the body can be changed to the lateral or supine position. A shift to the sitting position, however, results in a compressing force at the epicardial or pericardial surface which can flatten a soft polyethylene catheter, but not the stiffer Polyvinyl catheter. Such maneuvers were carried out in 6 patients without complication.

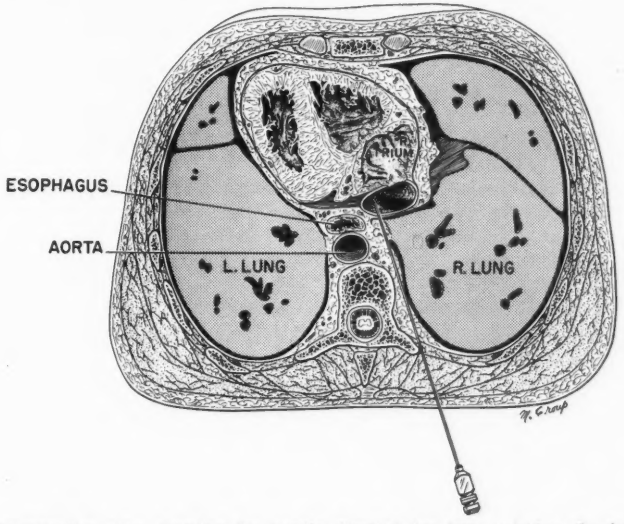


Fig. 1*C*.—Cross-section view showing the needle directed too low, entering the inferior vena cava or right atrium.

The theoretical dangers of transthoracic left atrium puncture are many, but no serious consequences have resulted in this series of cases. Although the pericardial cavity commonly became contaminated with blood, actual tamponade effects occurred in only 1 patient, when the base of the aorta was entered directly from the pericardial cavity. In another patient, when the aorta was entered from the atrium, no tamponade or other complication occurred.

It appears that the likelihood of complications is small if the needle is directed accurately to the left atrium, and especially if the aorta and pulmonary artery are avoided (Figs. 1 and 2). The use of fluoroscopy as a guide seems to be helpful, the left atrium being assumed to occupy the central area

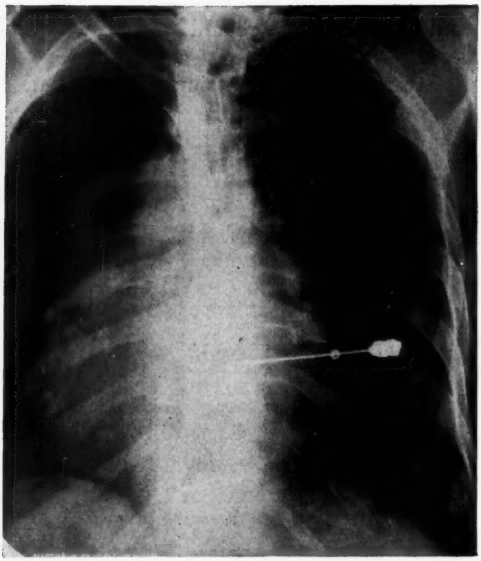


Fig. 2*A*.—Roentgenogram showing Patient J.F. (No. 42) in prone position, as seen at fluoroscopy, with needle placed in left atrium. The marker indicates the point of skin entry, on the right side.

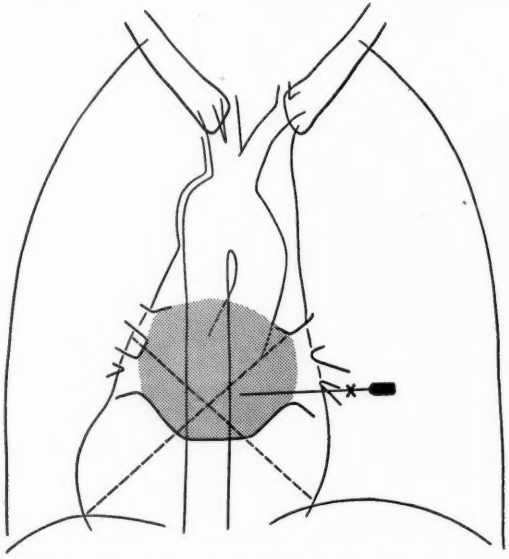
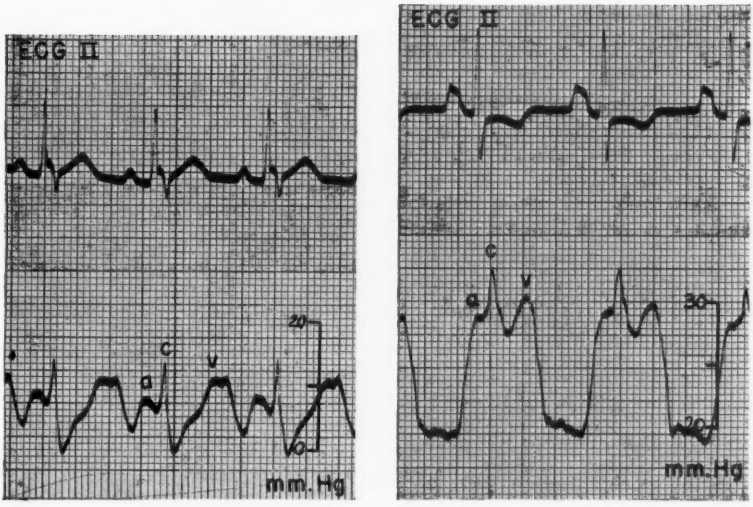


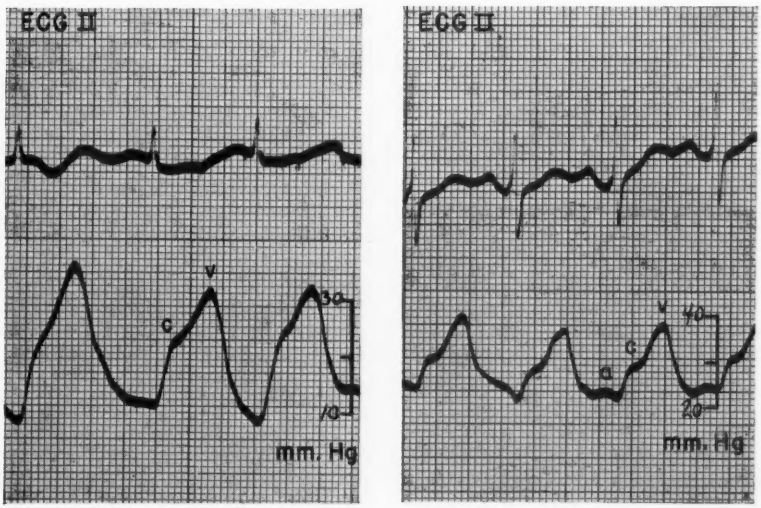
Fig. 2*B*.—Diagram indicating shaded area as the position of the left atrium on the heart shadow. The needle is directed from the point of skin entry toward the intersection of imaginary lines at the center of the heart shadow.



A. *B.*

Fig. 3.—*A*, Left atrial pressure recording in a heart with normal valves (obtained during right heart catheterization via a patent foramen ovale).

B.—Left atrial pressure recording in a heart with a "tight" mitral stenosis and no insufficiency.



C. *D.*

Fig. 3.—*C*, Left atrial pressure recording in a heart with mitral stenosis and a severe degree of mitral insufficiency.

D, Left atrial pressure recording in a heart with "pure" mitral insufficiency.

(Fig. 3*A*, *B*, *C*, and *D* is reproduced by permission of the Editor, *Annals of Surgery*).

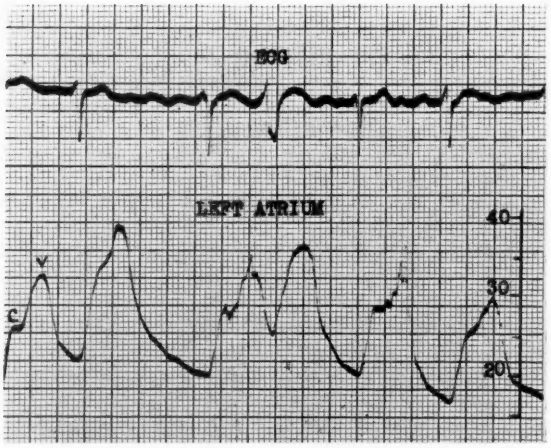


Fig. 4*A*.—Left atrium pressure recording in Patient C.H. (No. 45), mitral stenosis and insufficiency, showing the immediate ascent of the V-wave following the C-wave, due to the regurgitant pulse.

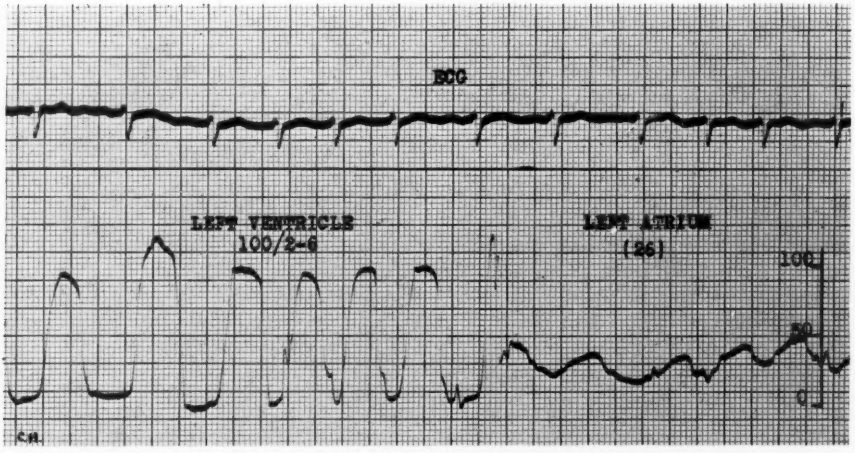


Fig. 4*B*.—Patient C.H. (No. 45), showing the recording during withdrawal of the catheter through the mitral valve. The pressure difference during diastolic periods is due to stenotic obstruction at the valve.

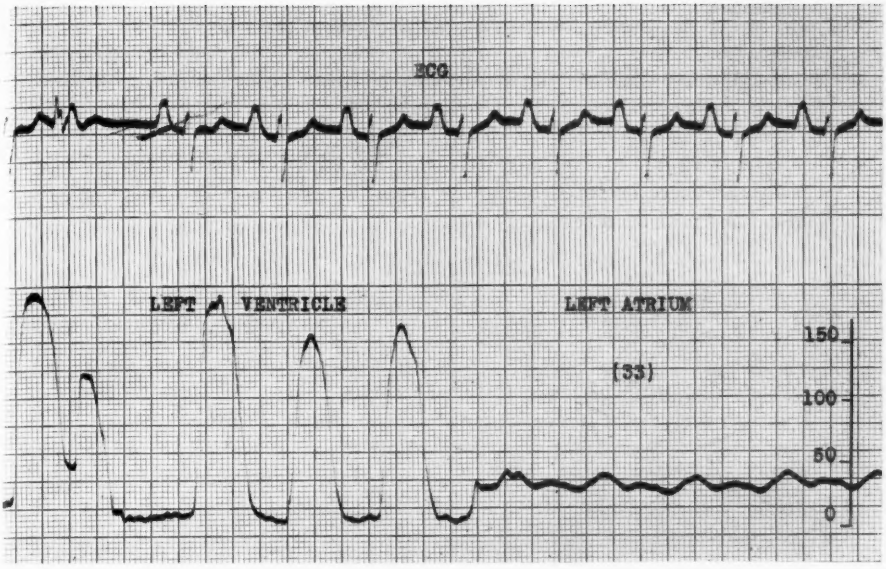


Fig. 5*A*.—Patient M. C. (No. 40), mitral and aortic stenosis with slight mitral and aortic insufficiency. Recording of pressure during catheter withdrawal through the mitral valve, showing diastolic phase pressure difference between atrium and ventricle, due to mitral stenotic obstruction.

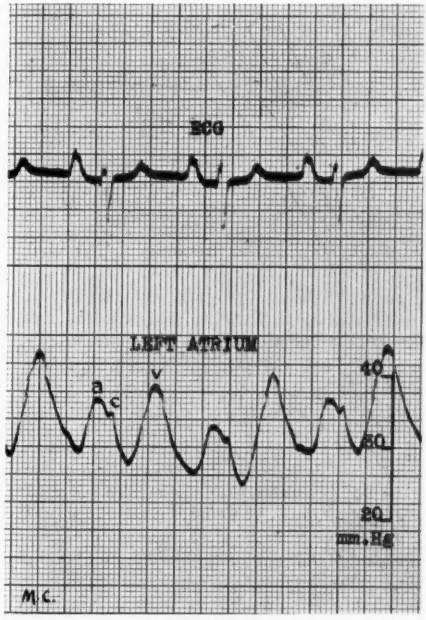


Fig. 5*B*.

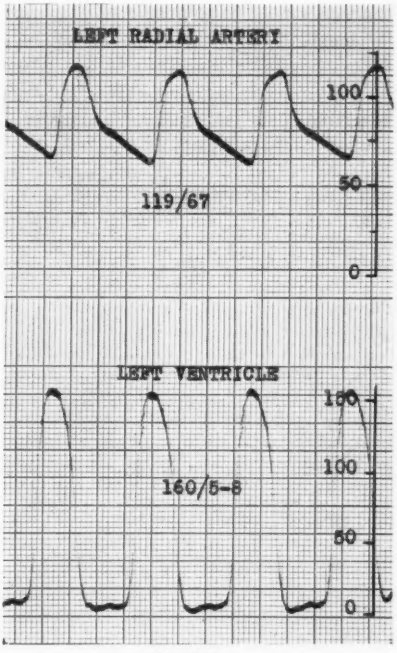


Fig. 5*C*.

Fig. 5*B*.—Patient M. C. (No. 40). The left atrial pressure curve has a tall V-wave, but this is less significant of a mitral regurgitant pulse because of the deep fall in pressure following the C-wave.

Fig. 5*C*.—Patient M. C. (No. 40). The recording demonstrates the drop in systolic pressure between ventricle and artery, due to stenotic obstruction at the aortic valve.

of the heart silhouette, and the needle being advanced toward an imaginary point in the exact center of the two-dimensional image. With the point of entry into the skin as used here, and an angle toward the left of 25 degrees, both the bony cage and the lung were avoided with the needle. No case of pneumothorax therefore occurred. The transthoracic method of heart catheterization has so far been relatively safe.

CONCLUSIONS

1. Sixty-one pressure recordings were obtained at transthoracic left atrium puncture for diagnosis in 58 patients.

2. Left heart catheterization was performed through the left atrial puncture needle in 52 of these tests. Diagnostic interpretations were compared with a series of observations at 26 later heart valve operations and 8 autopsies.

3. Only one major disagreement occurred in these comparisons. The pressure tests therefore appear to be reliable in the preoperative diagnosis of heart valve lesions.

4. Further precision should nevertheless be sought in the future by measurement of cardiac output, which is a major factor influencing the quantitation of the various pressure abnormalities.

5. No fatalities nor lasting sequelae resulted from the tests. There were only 3 minor complications, occurring in 3 of the first 14 patients of the series.

REFERENCES

1. Björk, V. O., Malmstrom, G.. and Uggla, L. G.: Left Auricular Pressure Measurements in Man, Ann. Surg. **138**: 718, 1953.
2. Björk, V. O.: Direct Pressure Measurement in the Left Atrium and Left Ventricle and the Aorta, Acta chir. scandinav. 107, Fasc. **5**: 468-476, 1954.
3. Fisher, D. L., Childs, T. B., Ford, W. B., and Kent, E. M.: Left Atrium Pressure Pulses in Mitral Valve Disease, Program of the Scientific Sessions, Am. Heart Assoc., April, 1954.
4. Kent, E. M., Ford, W. B., Fisher, D. L., and Childs, T. B.: The Estimation of the Severity of Mitral Regurgitation, Ann. Surg. **141**: 47-52, 1955.

DISCUSSION

DR. WILLIAM S. BLAKEMORE, Philadelphia, Pa.—I want to congratulate Dr. Fisher on the work which he has pioneered in this country, and this paper in which he has pointed out the diagnostic possibilities of this test. While working in Professor Crafoord's clinic with Dr. Björk and Dr. Malmstrom, we were able to develop an original method of catheterization of the left side of the heart. Later, Dr. Schnabel and I further modified the procedure and we now use three small catheters.

The first catheter is inserted through a No. 18 thin-walled needle into the left auricle and through the mitral valves into the left ventricle. A second catheter is inserted into the auricle and a third is inserted via a percutaneous needle puncture into the femoral artery and passed to the aortic arch near the valve. We have used this procedure in 14 patients who were poor operative risks and in whom other routine studies for evaluation of their cardiac disease had not proved the diagnosis. There is some risk to this procedure and Dr. Björk had one death in the series he studied, and we have had one death in this series.

In a patient with pure aortic stenosis a ventricular pressure was recorded of 160 mm. Hg and an aortic pressure of 70 mm. Hg. The diagnosis of aortic stenosis was confirmed

at operation. Interestingly enough, when the cardiac rate changes in these patients the pressure gradient across the valve decreases if the cardiac output remains the same. In a patient with pure mitral stenosis, it has been noted that the pressure gradient is maintained throughout ventricular diastole.

The first sound is markedly accentuated in mitral stenosis, and Dr. Schnabel has attributed this to the closure of the valves during the steep rise of the ventricular pressure. The aortic curve does not coincide exactly with the ventricular curve in the time axis because the tip of the catheter was located in the arch rather than at the aortic valves.

In another patient with mitral regurgitation and no mitral stenosis, there was a rise in the left auricular pressure during ventricular systole, and an insignificant pressure gradient across the mitral valve during ventricular diastole.

A delayed rise in the aortic pressure with the catheter placed at the level of the aortic valve may be due in some degree to aortic stenosis. Withdrawal curves with one catheter recording the pressure in the left ventricle, and another in the left auricle, show that as the left ventricular catheter is withdrawn into the auricle the two pressure curves can be superimposed, confirming the accuracy of this type of recording. It would therefore seem that this is a very reliable diagnostic method for determining mitral and/or aortic stenosis. We have, because of the risk involved, used this procedure only when other methods of cardiac evaluation have not provided a precise diagnosis in patients who are poor operative risks.

DR. CLARENCE CRAFOORD, Stockholm, Sweden.—I am sorry I did not have a program of this meeting before I left Stockholm, as I wanted to see if there was something I wished to discuss, and as a result I have no slides. Dr. Fisher's paper and Dr. Blakemore's discussion are interesting to me, as this method of recording was first carried out in my service. We had a few cases where the clinical symptoms led us completely astray in deciding whether insufficiency or stenosis was predominant, and for that reason this method was developed. I want to correct one thing Dr. Fisher said; he said that we did not use fluoroscopy to judge how the needle was introduced, but we always control this with fluoroscopy and, if necessary, x-ray photography to see if the needle is in the correct position.

I think we have had approximately another 100 cases which have been explored; the total number is about 200. We had one death. That patient never should have been subjected to this procedure. We were unable to identify a rather large shadow posteriorly, which had been interpreted as a large auricle, but it proved to be an aneurysm at the base of the aorta which was passed by the needle. The patient went into shock because of intrapericardial bleeding. A successful attempt was made to suture, but the patient did not recover. We had 1 case of rather severe intrapleural bleeding in this series which necessitated multiple aspirations and streptodornase-streptokinase treatment. We do not know the exact cause of this bleeding, but the patient recovered from this complication and was operated on for mitral stenosis about two months afterward with complete success and without complications.

Otherwise, there have been some minor complications such as small pneumothoraces, some small bleeding in the pleural space, which have not necessitated any treatment. There was recently, however, one case of rather massive intrapericardial bleeding which necessitated a couple of aspirations of the pericardium. When we started the investigation only one thin catheter was passed through the needle. Attempts to use heavier needles in order to pass thicker catheters have been abandoned, because of the greater risk of after bleeding. It is always easy to pass the catheter into the auricle and through the mitral valve into the left ventricle. We have not succeeded in more than 40 per cent of the cases in passing the catheter into the aorta. We have also used two catheters in order to obtain simultaneous pressure curves in the ventricle and auricle, or either of them and the aorta. On one occasion when we started to take the catheters out, they were stuck. This was a case in which predominant stenosis or incompetence had been discussed. The

curves showed that it was a definite mitral stenosis. We had to take the patient to the operating room, and at operation, when successful commissurotomy was performed, we found that the two catheters had made a knot of which any seaman would be proud.

DR. A. HIMMELSTEIN, New York, N. Y.—I should like to continue this discussion basing it on our experience with 8 transbronchial needlings of the left auricle. Our experience has been with mitral disease, and I should like to show some slides to validate the recordings in the operating room as compared to recordings with intact chest wall.

This slide is a tracing made in the operating room by an apparatus which enables us to use identical sensitivity manometer recording systems and to superimpose them so that we get a tracing which is very similar to the one in Wiggers' textbook, which Dr. Fisher showed. In this patient with mitral stenosis, needles were placed in the left auricle, left ventricle, and aorta. We see here the ventricular tracing, the aortic tracing, and the auricular tracing, and the hallmark of any stenosis is very obvious here in the gradient of pressure between auricle and ventricle during diastole. This is a patient who had been operated on once before for mitral stenosis. I would interject here that this is not a recurrence, since when we spoke to the surgeon who did the first procedure we realized that this valve had not been opened.

This slide shows a transbronchial puncture in the same patient, done preceding operative intervention. It shows a slightly damped ventricle tracing. The needle is an 18 gauge thin-walled one, 50 cm. long, that goes through the bronchoscope, and a catheter is passed through this into the ventricle. The catheter is similar to the one Dr. Fisher uses. The auricular tracings are taken through the needle and are recorded with a much better frequency response. We have a pressure in the auricle which is elevated, and it brings out the marked pressure gradient during diastole and the fact that these tracings are very similar both in contour and absolute value to what we get in the operating room.

This slide shows the postoperative tracing in this patient done in the operating room. One should be able to tell when a good opening of the mitral valve has been secured. You get rid of the gradient which here (during all of diastole) has been reduced to almost zero, and at the end of diastole, to zero.

We too have had the problem of the diagnosing of mitral insufficiency, and in this context I show the next case.

(slide) These are the transbronchial tracings, the auricular tracings, and the ventricular tracings, to show that this patient had almost no diastolic gradient and therefore no significant mitral stenosis by our criteria. However, on the insistence of our medical colleagues, and some suspicion of our own—this is the first needling of the auricle we did—we proceeded to operate on this patient.

(slide) Here is the tracing made in the operating room, showing that the gradient of mitral stenosis is minimal, and when a finger was inserted into this patient's auricle, the problem was largely one of insufficiency with no stenosis.

I think it is extremely difficult to diagnose mitral insufficiency even by tracings, and we have seen rather significant mitral insufficiency which was not evident on tracings from the auricle.

DR. CHARLES P. BAILEY, Philadelphia, Pa.—We, too, have been stimulated by the report from Dr. Crafoord's clinic by Dr. Björk and his associates, and with the direct help of Dr. Donald Fisher we were able to start upon a similar program some months ago. We would like to take exception to Dr. Himmelstein's saying that tracings taken in the operating room are entirely reliable because, at least with our equipment and technique, there has been a great difference between them and the preoperative tracings obtained by this method. We have come almost to discard measuring the pressure in the operating room for that reason. Undoubtedly the discrepancy is related to a diminished stroke output during the course of anesthesia and surgery.

In valvular disease, one frequently is hard put to decide what lesions are present, what lesions are important, which to attack first, and which may safely be left alone. This method of investigation has been of great value to us, although we have carried

it out in only 29 patients. There was one death in this series, occurring a day after left heart catheterization. This was a patient with severe insufficiency of the mitral valve. At post-mortem, the death did not seem in any way to be related to the procedure. We have had several patients with fluid in the pericardium, which was sanguineous. We are pretty sure that we know all about this complication because we have limited this study to patients who were about to be operated on the next day or the day after.

There was one instance of inadvertent puncture of the aorta, due to misdirection of the needle. Some degree of hematoma of the mediastinum and periaortic region resulted but it was in no way serious.

Recently, Dr. Goldberg, our physiologist, has been doing simultaneous right heart catheterization and left heart catheterization, thus being able to measure not only the cardiac output but the pressures on the left side, and also the condition of the tricuspid valve, pulmonary capillary resistance of the lung, and things of that sort. We feel that the procedure is quite safe and reasonable, and that it should be employed more often, especially when one is dealing with suspected multivalvular disease. We feel that Dr. Crafoord and his group, and Dr. Fisher, who took the lead in this country, have certainly given us a very worth-while tool for evaluation of these problems.

DR. OSLER ABBOTT, Emory University.—I admit there is a difference between tracings taken in the laboratory and in the operating room, but these are differences only of degree and both tracings can be usefully correlated. We have been studying the pressures in the pulmonary artery, pulmonary vein, and auricles at the time the chest is open. These have been compared to preoperative cardiac catheter studies. The tracings obtained at the time of open chest are variable according to three basic factors: first, the intrabronchial pressure produced by the ventilatory pressure of the anesthesia machine; two, the drugs acting at the time the pressure is taken; and three, the patient's position. Pressures at cardiac catheterization and at the operating table will parallel each other in their contour. An interesting finding in this study of left heart pressures at the table, with controlled endobronchial ventilatory pressures ranging from zero to 5 mm. Hg is that the pulmonary venous pressures are negative only during the zero or expiratory phase, and then they may have a negative characteristic of no more than 1 to 3 mm. Hg. When the endobronchial ventilatory pressures are increased to (1) zero at 10 mm. Hg and then to (2) zero to 20 mm. Hg, the pressure readings in the pulmonary vessels normally parallel the endobronchial ventilatory pressures. This response varies somewhat in different pulmonary and cardiovascular disease states.

(slide) It was interesting to us in looking over a respirator that had a negative phase of expiration to find that a negative pressure phase appears in the pulmonary artery. This suggests that there may be a phase of backflow of blood in patients in the lateral position from the pulmonary arterial bed of the uppermost or exposed lung over to the contralateral lung. This is a significant degree of negative intra-arterial pressure. This response is not present in patients with significant degrees of pulmonary arteriosclerosis.

(slide) In the pulmonary vein a marked degree of constant negative pressure exists at all times when a negative-phase respirator is used. When the left auricle is studied directly, the intra-auricular pressure exactly parallels the venous pressure. In the open chest, when using a biphasic respirator anesthesia machine (with a negative expiration component), we have found constant recordings of negative pressures in the pulmonary arterial system during expiration and, at all times, in the pulmonary venous system and in the auricle. These are the findings in the essentially normal cardiovascular patient.

I think this brings up a considerable concern about biphasic pressure respirators for open thoracotomy. I think it also emphasizes the remark that the ventilatory pressure and the other two factors mentioned have a great deal to do with the paralleling of intraluminal pressure studies obtained in the laboratory and in the operating room.

DR. BERNARD BERCU, St. Louis, Mo.—I should like to show a slide which indicates that our results are almost exactly parallel to his, and I can summarize our criteria by saying they are exactly the ones he presented.

We have had 30 cases in conjunction with Dr. Thomas Burford at Barnes Hospital and I think a few points of difference between our technique and Dr. Fisher's might be worth mentioning. First, we do not use the Trendelenburg position, but in all instances have the patient lying on his right side. We have not used the fluoroscope. This has made the procedure rather simple and has allowed us to get more freedom of movement. The left auricle has been entered in all instances. In 1 case, the left ventricle was entered first and in 2 cases the pulmonary artery first, but upon withdrawing the needle it could then be passed into the left auricle. After the auricle is entered, a polyethylene catheter is inserted through the needle. In passing the catheter through the auricle we have also had uniform success in passing it into the ventricle. In 4 of these cases we were unable to pass the catheter into the ventricle. These were in our first seven attempts. Since then we have had no difficulty.

With regard to passing the catheter into the aorta, we did not attempt to do this, as we felt that it added little to the clinical impression we obtained. We have had no serious complications and no incidence of bleeding or hemothorax. Interestingly enough, there was a small amount of serosanguineous fluid at operation within the next 24 to 72 hours. This has been a very tiny amount of fluid, and no noticeable puncture could be seen in the left auricle.

I think I might summarize by saying that it is our feeling that the procedure as described by Dr. Fisher is a simple one and very useful clinically, and that complicating it by adding additional factors would make it more complicated and bulky. We feel it can be done without such additional things.

DR. GEORGE W. B. STARKEY, Boston, Mass.—I should like to ask the author or any of the discussants of his paper how well their pre- and postoperative measurements of the gradient across the aortic valve, using the Björk method, have correlated with measurements of the gradient at the time of surgery, both before and after dilating the aortic valve.

I should like to congratulate Dr. Fisher for this excellent paper.

DR. DON L. FISHER, Pittsburgh, Pa. (Closing).—The only answer I have to this last question is to cite 1 case, in which there was calcified aortic stenosis. At a preoperative left heart catheterization test, the systolic pressure decreased by 70 mm. Hg at the aortic valve. At operation, the same pressure drop was determined by passing the catheter down the aorta from above, through the opening of the aortic incision, and then a systolic pressure drop of 40 was obtained at the valve. I was not able to make postoperative recordings, unfortunately. I do not have other such comparative tests regarding aortic stenosis.

We have, however, found in mitral stenosis a good qualitative correlation between preoperative measurements and tests at operation. The test conditions appear to affect the measurements. For example, in one case of mitral stenosis there was a diastolic pressure drop of 39 mm. Hg at the mitral valve, preoperatively. This patient was insufficiently sedated, and was apprehensive. At the time of operation, the pressure drop was 20 before finger fracture of the valve, and 9 afterward, with less than an ideal result on palpation of the valve.

Cardiac output measurements, study of the heart rate, and so on, will be helpful in the use of pressure recordings. Body position may have little effect on the contour of the left atrial pressure curves. We studied this in preoperative tests by withdrawing the needle over the catheter, leaving only the soft catheter in place. Change to various lateral, sitting, and supine positions then was found to cause no significant change in contour of the left atrial pressure curves. Exercise in the supine position caused elevation of left atrial pressure with increase of any regurgitation effect.

A LABORATORY AND CLINICAL EVALUATION OF OPERATIONS FOR CORONARY ARTERY DISEASE

DAVID S. LEIGHNINGER, M.D. (BY INVITATION)
CLEVELAND, OHIO

DURING the past twenty-three years Beck and his associates[1] have established the principles of improving the blood supply to the heart. These are (1) the even distribution of blood by intercoronary arterial communications, (2) elevation of the coronary sinus pressure, and (3) the addition of blood from outside sources by (a) extracoronary communications and (b) arterialization of the coronary sinus.[1]

In recent years the importance of intercoronary arterial communications as a mechanism of benefit has become more apparent. An appreciation of this importance depends upon an understanding of the mechanisms by which patients die from coronary artery disease. There are two types of death.[1] One type follows extensive degeneration of the myocardium. The myocardium gives way and heart failure develops. This type of death has been called *muscle death*. The other type of death follows a break in the coordinated mechanism of the heartbeat. The pumping action of the heart is destroyed and death occurs immediately. This type of death has been called *mechanism death*. The hearts of patients who die a mechanism death may show little or no degeneration of the myocardium. No recent lesion may be found in either the arteries or muscle.[2, 3] Presumably these hearts were capable of continued function had not a break in mechanism occurred. Analysis of 950 deaths from coronary artery disease reported by Yater and co-workers[3] shows that about 90 per cent died from a break in mechanism and about 10 per cent died from heart failure.[1] Attention is directed to this large group of patients who develop a break in the coordinated mechanism because something can be done to prevent this break.

The hearts in many of these victims are capable of continued function provided the break does not occur, whereas in the other group of patients nothing can be done to restore myocardium after it degenerates into scar.

The hearts in which the coordinated mechanism is lost are electrically unstable. Experimentally a heart can be made unstable and the normal mechanism can be readily lost.[4] Fig. 1 is an electrocardiographic tracing which was taken from the surface of the left ventricle of a normal dog heart. The electrode

From the Western Reserve University School of Medicine and the University Hospitals.

This work was supported by grants from the United States Public Health Service and the Cleveland Area Heart Society, and was performed in the laboratory of Claude S. Beck.

Acknowledgment is made for the assistance of Drs. J. J. Perez Alverez, Mexico City, Mexico; M. Wilson, Bristol, England; H. D. Pouw, Djakarta, Indonesia; and S. Oudkerk, Bennekom, The Netherlands.

Read at the Thirty-fifth Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, N. J., April 24 to 26, 1955.

was placed directly on the epicardium. These tracings showed uniform electrical potential during diastole and during the repolarization phase of systole. The "S-T segment" of the complex is recorded during the repolarization phase. If a portion of the ventricle is made anoxic by ligation of its arterial blood supply, then the electrocardiogram is altered over this part of the heart.

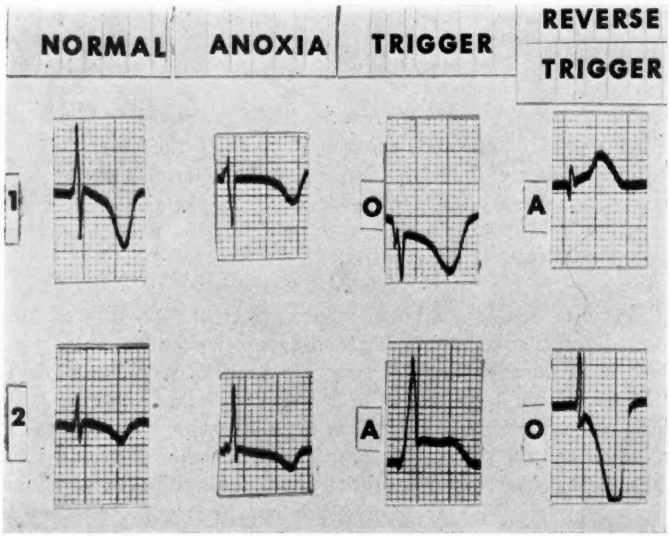


Fig. 1.—Epicardial electrocardiographic tracings from a dog heart. The complexes in line 1 were taken from the same location in the descending area. The complexes in line 2 were taken from the same location in the circumflex area. *O* = oxygenated area; *A* = anoxic area.

The S-T segment is elevated over the entire anoxic area and the S-T segment is depressed over the adjacent oxygenated area (Fig. 1). This indicates that during diastole the anoxic area is electrically negative as compared with the oxygenated myocardium. The area rendered anoxic by arterial ligation is sharply demarcated from the pink oxygenated area. This is readily visible. The transition between blue and pink muscle is an extremely narrow zone. Measurements from one zone to the other were 10 to 20 millivolts. This indicates electrical imbalance between the blue and pink areas of the heart and produces what might be referred to as an electrically unstable heart because it might fibrillate. It is interesting to note that a uniformly anoxic heart such as is produced by clamping the intratracheal tube for periods of six to eight minutes is a relatively stable heart. In this type of experiment the heart becomes extremely blue and it does not fibrillate. Approximately 50 such experiments were performed by me and none of these hearts fibrillated. At the suggestion of Dr. Beck a reverse trigger was made by injection of red blood into the descending ramus or the circumflex ramus of the left coronary artery. This produces a pink trigger in a blue heart

and the epicardial leads were negative over the blue areas compared to the pink area. The same difference in electrical potential was produced except that the alterations were reversed. Such hearts became unstable and some of them went into ventricular fibrillation. These experiments demonstrate that blue muscle in contact with pink or pink muscle in contact with blue produces currents which can make the heart fibrillate. The dangerous area is in the plane of contact between the two areas. This danger can be reduced by diminishing the difference in oxygen tension between the two areas and this can be accomplished by establishing arterial communications between the two areas. The importance of intercoronary communications is obvious. This demonstration by experiment can be applied to the vast majority of people who die from coronary artery disease and it explains death occurring in hearts that show little or no disease.

The quantity of blood necessary to prevent a break in mechanism is not large. Eckstein and associates[5] have shown that in every experiment 4 c.c. of additional blood per minute delivered to the circumflex area prevented death within one hour following circumflex artery ligation. In control dogs the mortality within one hour was 70 per cent following circumflex artery ligation.

Intercoronary arterial communications can distribute blood evenly to all parts of the myocardium and thus maintain electrical stability. Ninety-one per cent of normal human hearts have no intercoronary communications demonstrable by injection studies.[6] In normal dogs 90 per cent have insufficient intercoronary communications to prevent death from circumflex artery ligation and 70 per cent have insufficient intercoronaries to prevent death from either descending or right coronary artery ligation.

Permanent intercoronary arterial communications can be produced by operation. Various stimuli, or combinations of stimuli, have been employed at operation and reported by Beck.[1]

A quantitative evaluation of intercoronary arterial communications can be made by the Mautz-Gregg backflow method.[7] The circumflex ramus of the left coronary artery is ligated near its origin and cannulated distal to the ligature (Fig. 2). The artery is perfused with arterial blood by way of a cannula in the carotid artery. This supplies oxygenated blood to the circumflex area of the myocardium except when a backflow measurement is made. To measure the backflow the perfusion is stopped and a side arm of the cannula is opened. Blood from other coronary arteries flows across whatever intercoronary communications may be present into branches of the circumflex artery. From these branches it flows in a retrograde direction out of the artery through the cannula and the side arm. It is collected for a period of thirty seconds and measured. The backflow is expressed as cubic centimeters per minute. Several measurements are made and the results averaged. Backflow measurements are always made with the mean aortic blood pressure maintained at 100 mm. mercury. The greater the number and functional

capacity of the intercoronaries, the greater will be the backflow. After backflow measurements have been made the circumflex perfusion is stopped. The only blood that reaches the circumflex area of myocardium is that which flows across intercoronary communications. Electrocardiographic tracings, using lead aV_R, are taken for a period of nine or ten minutes after the perfusion is stopped. Electrocardiographic changes are graded according to the criteria of Eckstein and Leighninger.[8] Changes of "two plus" or less are arbitrarily considered as showing evidence of protection. Electrocardiographic evidence of protection correlates well with the one-hour mortality of 70 per cent from circumflex artery ligation as found by Eckstein,[5] but shows greater protection than was found in the long-term mortality of 90 per cent from circumflex artery ligation as reported by Leighninger and Dalem.[9]

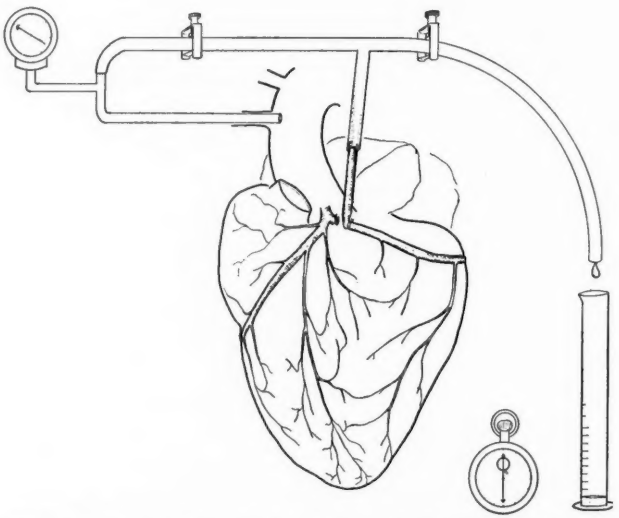


Fig. 2.—Mautz-Gregg backflow method. Circumflex artery ligated and distal segment cannulated. Cannula perfused by way of the carotid artery. Blood pressure recorded from the carotid cannula. Side arm from circumflex cannula is clamped during perfusion. When carotid cannula is clamped and side arm opened backflow blood flows against atmospheric pressure. The end of the side arm tube is at the level of the heart. Backflow blood is collected for a period of 30 seconds and expressed as backflow in cubic centimeters per minute.

Backflow determinations have been made on a group of 67 normal control dogs.* The average backflow was 3.8 c.c. per minute. The range was from 0.4 c.c. per minute to 21.0 c.c. per minute. The median backflow was 3.0 c.c. per minute. Twenty-five per cent of the normal dogs showed electrocardiographic evidence of protection.

Backflows from normal dogs were compared with backflows obtained from 41 Beck No. 1 operated dogs. Operation consisted of abrasion of the epicardium and pericardium, partial coronary sinus ligation, application of

*Eckstein[10] studied 41 of these normal dogs.

0.2 Gm. powdered asbestos, and grafting of the abraded pericardium to the surface of the heart. Backflow determinations were made from three weeks to one year after operation. The average backflow was 8.5 c.c. per minute. The range was from 0.6 c.c. per minute to 26.0 c.c. per minute. The median was 7.0 c.c. per minute. Sixty-five per cent of the dogs showed electrocardiographic evidence of protection. The backflows for the Beck No. 1 operated dogs are significantly different from those of the control dogs. The probability of such a difference occurring by chance alone is greater than one chance in 1,000. Five chances out of 100 is usually considered statistically significant.

The Beck No. 1 operation added an average of 4.7 c.c. per minute of blood to the circumflex area by way of intercoronary communications. Operation stimulated the growth of these communications. Since they persisted for at least one year after operation they are considered permanent.

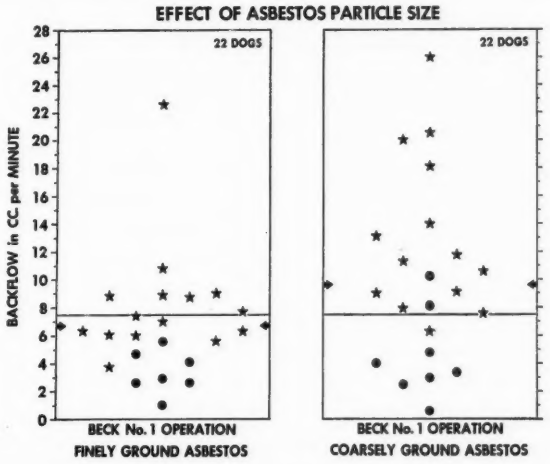


Fig. 3.—Effect of asbestos particle size on the No. 1 operation. Each dot or star represents the backflow from one dog. A star indicates presence of electrocardiographic evidence of protection. The horizontal line is through the median for the entire group of dogs. The average backflow for each group is indicated by arrows.

The 4.7 c.c. per minute of blood added by operation is a significant quantity of blood. It prevents electrical instability in hearts following coronary artery ligation. It was responsible for lowering mortality to 26.6 per cent following descending artery ligation in dogs protected by the No. 1 operation. Mortality from descending artery ligation was 70 per cent in normal dogs.[1] This quantity of blood was responsible for preserving the viability of myocardium following artery ligation as shown by the reduction by 70 per cent of infarct size in protected dogs as compared with normal dogs.[11]

The effect of coronary sinus ligation on the Beck No. 1 operation was studied. In all dogs the pericardium and epicardium were abraded and 0.2 Gm. powdered asbestos applied to the surface of the heart. In 5 dogs

the sinus was not ligated and backflows averaged 8.1 c.c. per minute. In 5 dogs the sinus was completely ligated near its ostium. The average backflow was 5.5 c.c. per minute. In 41 dogs the sinus was partially ligated. The average backflow was 8.5 c.c. per minute. The rationale for including partial sinus ligation as a step in the No. 1 operation includes the results of acute studies by Eckstein and associates.[12] Elevation of the coronary sinus pressure followed immediately by circumflex artery ligation lowered the one-hour mortality from this artery ligation from 70 per cent in control dogs to 35 per cent in the dogs with sinus pressure elevation. The immediate benefit thus demonstrated probably accounts for at least some of the early postoperative benefit noted by some patients following operation.

Fig. 3 compares the effect of asbestos particle size on the Beck No. 1 operation in a group of 44 dogs. The asbestos used in one group was finely ground in a mortar and sifted through several layers of gauze. In these dogs the average backflow was 6.7 c.c. per minute. The asbestos used in the other group of dogs was either lightly ground in an ordinary household food grinder or used just as it was received from the manufacturer. The average backflow for these dogs was 9.1 c.c. per minute. The coarser-ground asbestos is now standard for use in patients.

Pilot studies were carried out in a group of 161 dogs following a variety of operative procedures.

Forty-three dogs were studied to determine the effects of the individual components and combinations of components of the Beck No. 1 operation. The average backflow following complete sinus ligation was 11.0 c.c. per minute. McAllister and Leighninger[13] reported a 4.2 per cent mortality following complete sinus ligation in dogs. One of Dr. Beck's patients had complete sinus ligation alone. This patient received an excellent result. It has been shown previously, however, that, in combination with abrasion and application of asbestos, partial sinus ligation gave better results than complete sinus ligation. Postoperative morbidity in dogs following complete sinus ligation was slightly higher than following partial sinus ligation, either alone or in the complete Beck No. 1 operation. In 7 dogs the sinus was ligated to either 2 or 3 mm. The average backflow was 7.0 c.c. per minute. In 7 dogs the epicardium was abraded and backflows averaged 6.9 c.c. per minute. In 5 dogs 0.2 Gm. powdered asbestos was applied to the surface of the heart. The average backflow was 5.7 c.c. per minute. In 5 dogs the heart was abraded and asbestos applied. The average backflow was 8.1 c.c. per minute. In 5 dogs the heart was abraded and the sinus ligated to 3 mm. Backflow averaged 10.8 c.c. per minute. In 5 dogs the sinus was ligated to 3 mm. and asbestos applied. The average backflow was 8.5 c.c. per minute. The average backflow for the 43 dogs studied was 8.5 c.c. per minute. Sixty-eight per cent of the dogs showed electrocardiographic evidence of protection. These studies show that each of the components of the No. 1 operation, either singly or in combination, stimulates the growth of intercoronary communications. The differences found in this

small series are not statistically significant. The effect of the components, when combined in the complete operation, however, is not necessarily the sum of the benefit from each.

Asbestos is a naturally occurring mineral composed mainly of silica and magnesium oxide, small amounts of aluminum and ferric oxide, and traces of other compounds. Its physical properties and chemical composition vary slightly depending upon where it is mined. Talc is similar in composition, but contains larger quantities of silica. It also varies in composition depending upon where it is mined. Pilot studies were made in a group of 49 dogs using each of ten varieties of asbestos or talc. Each differed slightly in

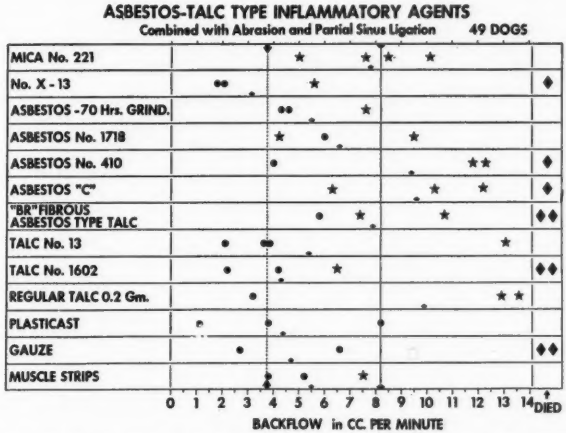

ASBESTOS-TALC TYPE INFLAMMATORY AGENTS
Combined with Abrasion and Partial Sinus Ligation 49 DOGS
MICA No. 221
No. X - 13
ASBESTOS -70 Hrs. GRIND.
ASBESTOS No. 1718
ASBESTOS No. 410
ASBESTOS "C"
"BR" FIBROUS ASBESTOS TYPE TALC
TALC No. 13
TALC No. 1602
REGULAR TALC 0.2 Gm.
PLASTICAST
GAUZE
MUSCLE STRIPS
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 DIED
BACKFLOW in CC. PER MINUTE

Fig. 4.—The effect of various irritants on the No. 1 operation. Each dot or star represents the backflow from one dog. A star indicates presence of electrocardiographic evidence of protection. Each diamond represents a postoperative death. The dotted vertical line indicates the average backflow for control dogs. The solid vertical line indicates the average backflow for Beck No. 1 operated dogs. The average backflow for each group of dogs is indicated by an arrow. The names in the left column are code names for various types of irritants.

chemical composition and physical properties. The backflows obtained, together with the averages for each group of dogs, are shown in Fig. 4. In each dog the heart was abraded, the sinus partially ligated, and the test substance applied to the surface of the heart. The average backflows varied from 3.1 c.c. per minute to 9.9 c.c. per minute. No one irritant was significantly better than another considering the small number of dogs in each group. Also tested with this group was a ground plastic, the application of one layer of surgical gauze sponge to the surface of the heart, and the application of isolated pectoral muscle strips to the surface of the heart. The rationale for the use of muscle strips is as follows. Coronary artery ligation is a powerful stimulus to the growth of intercoronary communications. This is thought to be due to both pressure differences between the occluded artery and nonoccluded vessels and probably to anoxia. In infarction there is muscle death. The products of degeneration are absorbed through the lymphatics of the myocardium. Perhaps some substance was present in the products

of degeneration which stimulated the growth of intercoronary communications. Products of degeneration of the muscle strips applied to the surface of the heart would be absorbed at least in part by the lymphatics of the myocardium and any stimulus present could exert its influence. The average backflow of 5.5 c.c. per minute obtained in this group of dogs indicates that if such stimuli were present their influence was not great. There were 9 postoperative deaths in the 49 dogs tested, a mortality of 18 per cent. The average backflow for the entire group of 40 dogs was 6.5 c.c. per minute. Fifty per cent of the dogs showed electrocardiographic evidence of protection.

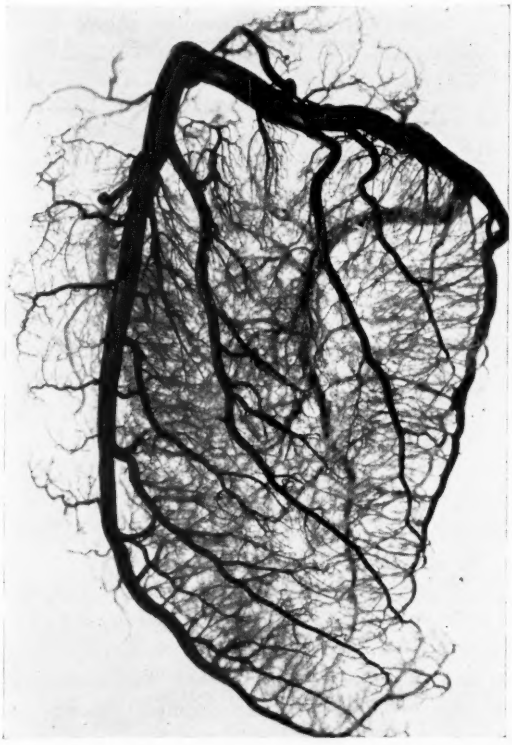


Fig. 5.—Plastic cast of coronary artery system in a normal human heart. Note the rich system of small vessels that were present within the substance of the myocardium. No intercoronary communications were demonstrated.

Most of the intercoronary communications which result from operation are present on the surface of the heart. There exists, however, a rich system of small arteries within the substance of the myocardium (Fig. 5). It was thought that the injection of a thin suspension of irritant into the myocardium might stimulate the growth of intercoronary communications between some of these vessels. Ten dogs were studied (Fig. 6). The average backflow was

6.98 c.c. per minute. Sixty per cent of the dogs showed electrocardiographic evidence of protection. The postoperative morbidity in these dogs was high. Many of the dogs showed disturbances in cardiac rhythm. It was concluded that injection of the irritant into the myocardium was no more beneficial than the standard operation. It was also considered more dangerous because of the morbidity and rhythm disturbances.

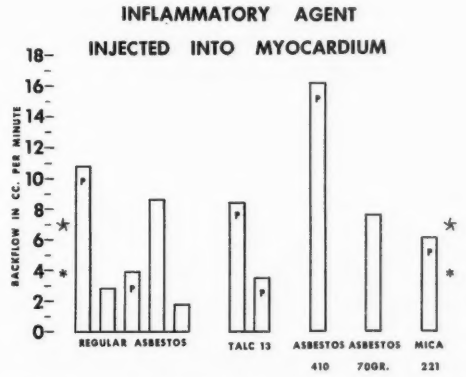


Fig. 6.—Each bar represents backflow from one dog. "P" indicates presence of electrocardiographic evidence of protection. The star indicates the average backflow for this group of dogs. The asterisk indicates the average backflow in control dogs.

Removal of the epicardium by chemical rather than mechanical means was tested. Fig. 7 shows the backflows obtained from dogs in which phenol, silver nitrate, or urea was tested. There were 6 postoperative deaths, 35 per cent mortality, in the group of 17 dogs. The postoperative morbidity was very high. The average backflow in the 11 surviving dogs was 5.2 c.c. per minute. Chemical removal of the epicardium, using the concentrations tested, was a dangerous procedure. By testing various concentrations of chemicals, a safe dosage might have been found. The results were not sufficiently encouraging, however, to warrant testing a large number of dogs in order to determine dosage.

Some surgeons have attempted to stimulate the growth of intercoronary and extracoronary communications by the application of an inflammatory agent to the surface of the heart without combining it with other mechanisms of benefit.[14] An example is the application of from 4 to 6 Gm. of talc to the surface of the heart. In a group of 22 dogs 4 Gm. of talc was applied to the heart. Several dogs developed compression scars with this dose of irritant. Two dogs died during the cannulation of the circumflex artery for study. These dogs were not protected by operation. Fig. 8 shows the backflows obtained from the remaining 20 dogs and compares them with normal dogs and to Beck No. 1 operated dogs. The average backflow was 4.9 c.c. per minute. Twenty-five per cent of these dogs showed electrocardio-

graphic evidence of protection. While application of talc alone elevated the average backflow above the average normal backflow by 1.1 c.c. per minute, the Beck No. 1 operation elevated backflows by 4.7 c.c. per minute.

Another attempt to improve the blood supply to the heart is the implantation of the internal mammary artery into the substance of the myocardium.[15] Fig. 9 shows the backflows obtained in 20 dogs in which the

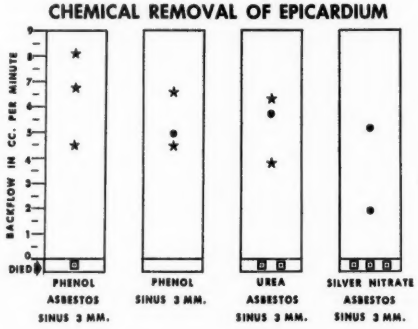

CHEMICAL REMOVAL OF EPICARDIUM
BACKFLOW IN CC. PER MINUTE
DIED
PHENOL ASBESTOS SINUS 3 MM.
PHENOL SINUS 3 MM.
UREA ASBESTOS SINUS 3 MM.
SILVER NITRATE ASBESTOS SINUS 3 MM.

Fig. 7.—Each dot or star represents backflow from one dog. Each square symbol represents a postoperative death. A star indicates the presence of electrocardiographic evidence of protection.

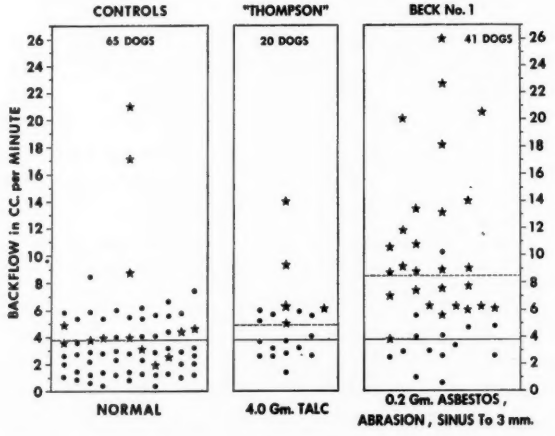


Fig. 8.—Backflows in normal dogs compared to backflows from Beck No. 1 operated dogs and dogs in which 4 Gm. of talc was applied to the surface of the heart. Each dot or star represents the backflow from one dog. A star indicates the presence of electrocardiographic evidence of protection. The solid horizontal line is through the average backflow for control dogs. The horizontal dotted line is through the average backflow for each group of dogs.

mammary artery had been implanted. No attempt was made to create a "physiologic need" by coronary artery occlusion. If a coronary artery is ligated or its lumen is reduced to less than 1 mm., growth of intercoronary communications is stimulated. Unless large quantities of blood were con-

tributed by the mammary artery graft it would be difficult to evaluate the function of the graft in the presence of these intercoronaries. In the dogs tested, backflows were obtained from both the circumflex and descending coronary arteries with the mammary graft both clamped and not clamped. Any difference in backflows with the graft clamped and not clamped would represent the amount of blood contributed by the mammary artery graft. Any elevation above normal backflows obtained with the mammary clamped would indicate the development of intercoronary communications as a result of the operative manipulation. The graft was occluded by thrombus in 5 of the 20 dogs. In none of the dogs was there a difference in backflow from either the descending or circumflex artery with the graft clamped or not clamped. In none of the dogs was the backflow significantly elevated above the average backflow obtained from normal dogs. In 20 dogs the average backflow from the circumflex artery was 3.81 c.c. per minute and from the

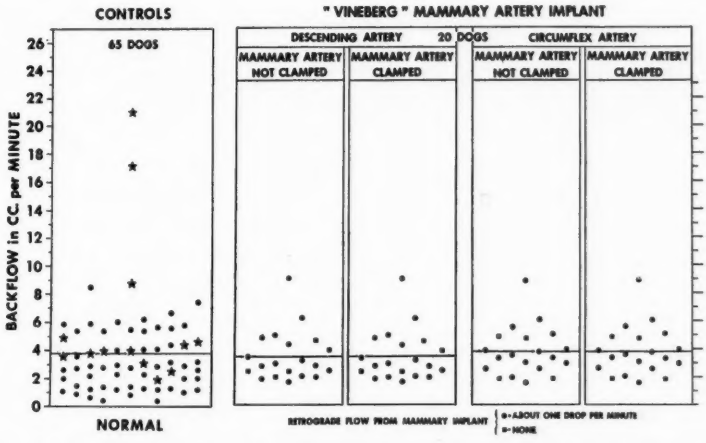


Fig. 9.—Backflows in normal dogs compared to backflows in dogs in which the internal mammary artery was implanted into the myocardium. Each dot, square, or star represents a backflow from one dog. A star indicates the presence of electrocardiographic evidence of protection. The square indicates absence of retrograde flow from the severed mammary implant.

descending artery was 3.5 c.c. per minute. None of the dogs showed electrocardiographic evidence of protection. After backflows had been obtained the mammary artery graft was cut between the heart and chest wall. In some grafts minute quantities of blood were seen to well up in the lumen of the artery on the heart side. This indicates that communications with coronary arteries were present but in such a small degree that the amount of blood was not measurable. The quantity was estimated as in the range of 1 drop per minute. It is concluded that under these conditions the internal mammary artery implant does not contribute significant quantities of blood to the coronary artery system.

The Beck operations for coronary artery disease have been applied to 240 patients during the past twenty years. Since January, 1954, only the present version of the No. 1 operation has been applied to patients. This consists of abrasion of the epicardium and pericardium; partial sinus ligation; application of 0.2 Gm. of powdered asbestos to the surface of the heart; and application of grafts of tissue, consisting of the abraded pericardium and of mediastinal fat, to the surface of the heart. Seventy-five patients have been operated upon during this seventeen-month period. There were two deaths from thoracotomy alone, before anything was done to the heart, a mortality of 2.6 per cent. There were three deaths in the early postoperative period, a mortality of 4.0 per cent. The total operative mortality was 6.6 per cent. During this time only two patients died subsequent to discharge from the hospital, a late mortality of 2.6 per cent.

The first-year late mortality has been calculated on the experience of 136 patients operated upon between 1935 and June, 1954. From time of discharge up to one year after operation there were 9 deaths, a mortality of 6.6 per cent. The second-year late mortality was calculated on the experience of 83 patients operated upon between 1935 and June, 1953. From one year up to two years subsequent to discharge there were 4 deaths, a mortality of 4.8 per cent. The total two-year mortality in patients treated surgically was 18.0 per cent (6.6 per cent operative mortality plus 6.6 per cent first-year mortality plus 4.8 per cent second-year mortality). In 88 patients who were acceptable candidates for sympathectomy for relief of angina pectoris but who were not operated upon for nonmedical reasons[16] there were 15 deaths the first year, 17 per cent, and 11 deaths the second year, 13 per cent, for a total two-year mortality of 30 per cent under medical treatment. Mortality under surgical treatment for a two-year period was 18 per cent. In the laboratory where conditions can be controlled and made comparable it has been shown that operation reduces mortality. Probably no two groups of patients are strictly comparable, even if alternate cases were selected for operation. However, these groups may have some comparable significance and, if so, it appears that a similar statement might be made concerning human patients.

In evaluating the results in patients, their pain and ability to work during the preoperative period have been compared to pain and ability to work in the postoperative period. Of 103 patients who were 6 months or more postoperative and who could be evaluated, 90 per cent have either no pain or marked reduction in pain and are better able to work. Some of these patients appear to be completely recovered from their symptoms. Others who have evaluated results in some of these patients have reported similar results.[1, 17]

These results place surgical operation in a favorable position for the treatment of coronary artery disease.

SUMMARY

1. The majority of deaths from coronary artery disease are due to unequal oxygenation of the myocardium resulting in electrical instability of the heart and a break in the coordinated mechanism of the heartbeat. These hearts were presumably capable of continued function had not the heartbeat been destroyed.

2. Intercoronary arterial communications, if present, can evenly distribute blood in the myocardium and prevent the development of electrical instability of the heart. As little as 4.7 c.c. per minute of additional blood to the myocardium beyond an occlusion can save life and save myocardium from destruction.

3. Operative manipulation of the heart stimulates the development of intercoronary arterial communications. Intercoronary communications resulting from a variety of operative manipulations have been evaluated by the Mautz-Gregg backflow method of study and the results presented.

4. There is no experimental evidence to indicate that medical therapy adds or subtracts a single drop of blood to ischemic areas of myocardium. The Beck No. 1 operation, however, provides the equivalent of a transfusion of 282 c.c. of additional blood per hour to the myocardium beyond the occluded circumflex artery.

5. The Beck No. 1 operation relieves symptoms to a significant degree in nine out of ten patients. The operative mortality is about 6.6 per cent. The trend of experience indicates that operation prolongs life.

6. Laboratory tests and clinical results place operation in a favorable position in the treatment of patients with coronary artery disease.

REFERENCES

1. Beck, C. S., and Leighninger, D. S.: Operations for Coronary Artery Disease, J. A. M. A. **156**: 1226, 1954.
2. Blumgart, H. L.: Relation of Effort to Attacks of Acute Myocardial Infarction, J. A. M. A. **128**: 775, 1945.
3. Yater, W. M., Welsh, P. P., Stapleton, J. F., and Clark, M. L.: Comparison of Clinical and Pathologic Aspects of Coronary Artery Disease in Men of Various Age Groups: A Study of 950 Autopsied Cases From the Armed Forces Institute of Pathology, Ann. Int. Med. **34**: 352, 1951.
4. Brofman, B., Leighninger, D. S., and Beck, C. S.: To be published.
5. Eckstein, R. W., Smith, G., Eleff, M., and Demming, J.: The Effect of Arterialization of the Coronary Sinus in Dogs on Mortality Following Acute Coronary Occlusion, Circulation **6**: 16, 1952.
6. Zoll, P. M., Wessler, S., and Schlesinger, M. J.: Interarterial Coronary Anastomosis in the Human Heart, With Particular Reference to Anemia and Relative Cardiac Anoxia, Circulation **4**: 797, 1951.
7. Mautz, F. R., and Gregg, D. E.: The Dynamics of Collateral Circulation Following Chronic Occlusion of Coronary Arteries, Proc. Soc. Exper. Biol. & Med. **36**: 797, 1937.
8. Eckstein, R. W., and Leighninger, D. S.: Chronic Effects of Aorta—Coronary Sinus Anastomosis of Beck in Dogs, Circulation Research **2**: 60, 1954.
9. Leighninger, D. S., and Dalen, J.: Revascularization of the Heart by Anastomosis Between Aorta and Coronary Sinus. The Beck II Operation—An Experimental Study, Ann. Surg. **140**: 668, 1954.
10. Eckstein, R. W.: Coronary Interarterial Anastomoses in Young Pigs and Mongrel Dogs, Circulation Research **2**: 460, 1954.
11. Beck, C. S., and Leighninger, D. S.: Operations for Coronary Artery Disease, Ann. Surg. **141**: 24, 1955.

12. Smith, G., Demming, J., Eleff, M., and Eckstein, R. W.: Further Studies on the Effect of Arteriovenous Fistulas and Elevations of Sinus Pressure on Mortality Rates Following Acute Coronary Occlusions, Circulation **6**: 262, 1952.
13. McAllister, F. F., and Leighninger, D. S.: Infarction of The Right Ventricle Caused by Multiple Coronary Vein Ligation, Circulation **1**: 717, 1950.
14. Thompson, S. A., and Plachta, A.: Fourteen Years' Experience With Cardiopexy in the Treatment of Coronary Artery Disease, J. THORACIC SURG. **27**: 64, 1954.
15. Bineberg, A., Munro, D. D., Cohen, H., and Buller, W.: Four Years' Clinical Experience With Internal Mammary Implantation in the Treatment of Human Coronary Artery Insufficiency Including Additional Experimental Studies, J. THORACIC SURG. **29**: 1, 1955.
16. Lindgren, I.: Angina Pectoris: A Clinical Study With Special Reference to Neurosurgical Treatment, Acta med. scandinav., supp. 243, **138**: 1, 1950.
17. Hellerstein, H. K.: Surgical Treatment of Acquired Nonrheumatic Heart Disease, J. M. A. Georgia **43**: 857, 1954.

(For Discussion, see page 432.)

TECHNICAL FACTORS WHICH FAVOR MAMMARY-CORONARY ANASTOMOSIS

With Report of Forty-five Cases of Human Coronary Artery Disease Thus Treated

Arthur Vineberg, M.D., and William Buller, M.D. (by invitation)
Montreal, Quebec

AT THE last meeting of this Association, the frequency of mammary-coronary anastomosis and mammary patency was questioned by one investigator. Because of this, it seemed advisable to review our knowledge concerning internal mammary artery implantation and the factors which play a part in preventing occlusion of the implanted artery.

This question has been studied by us for ten years, and our experimental data have been reported. Many hundreds of animals have been operated upon in testing one factor at a time. Today many of the factors responsible for occlusion of an implanted internal mammary artery are well known to us, as are some of the conditions which favor mammary-coronary anastomosis.

Factors which affect successful transference of a chest wall artery into the myocardium where it may function as an extracoronary artery are as follows: (1) the method of detachment of the internal mammary artery from the chest wall, (2) the method of implantation into the left ventricular myocardium, and (3) the condition of its myocardial environment.

In this article the technique of internal mammary artery implantation and the factors which seem to be responsible for keeping the artery open will be discussed together.

INCISION

With the patient in the right lateral position, the incision is carried from the mid-line anteriorly to the paravertebral line in the fifth interspace posteriorly. The fifth or sixth rib is removed extraperiosteally, depending on the preoperative fluoroscopic examination of the position of the apex. The rib below the apex is removed. If the thorax is resistant to lateral spread, it is best to cut the fifth or sixth rib posteriorly; otherwise, there is a tendency for the fifth or sixth costochondral junction to separate. Six cubic centimeters of 1 per cent procaine hydrochloride is injected into the pericardial sac and allowed to swish around during the preparation of the artery. The sternocostalis muscles are carefully removed from the fourth to sixth costal cartilages, which exposes the internal mammary artery and vein.

From the Department of Experimental Surgery, McGill University, and the Department of Surgery, Royal Victoria Hospital, Montreal.

Read at the Thirty-fifth Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, New Jersey, April 24-26, 1955.

PREPARATION OF INTERNAL MAMMARY ARTERY

(a) *Length of artery detached from chest wall.*—In our early work, the greatest number of arteries remained open and formed anastomoses when the internal mammary artery was freed from the chest wall between the fourth and sixth intercostal spaces. This principle is still followed, as we believe that when the artery is freed as far as the subclavian the risk of injury to the artery is increased and its blood supply may be jeopardized. When a long length of artery is freed, there is greater danger of angulation, kinking, and twisting. We have found many arteries occluded due to narrowing at a point of angulation caused by adherence to lung, sternocostalis muscles, etc. In many of these, the artery, although narrowed or occluded at the site of angulation, was completely patent beyond this point. Injection of the implant distal to the point of occlusion in such cases has demonstrated the presence of mammary-coronary anastomoses.

In human cases of coronary artery insufficiency, it may be necessary to obtain a longer portion of the internal mammary artery when: (1) there is a large anterior wall infarction; (2) the apex is in the seventh interspace; (3) the apex moves more than one interspace on respiration; or (4) there is left ventricular hypertrophy.

(b) *Excision of loose tissues in the area (sternocostalis, etc.).*—It is important to remove the sternocostalis muscles, fibrous pericardium and, in many instances, the mesial fat pad in order to prevent the artery from becoming adherent to these structures after implantation, with resulting angulation and occlusion at the point of adherence.

(c) *No-touch technique.*—It is hardly necessary to remind an audience such as this that direct handling of an artery may damage the intima, thus forming a nidus for clot formation. One research center reporting a series of blocked Vineberg implants, in addition to other technical errors, routinely placed bulldog clamps on the proximal end of the vessel! It is our custom never to grasp the artery with an instrument. After the sternocostalis muscles have been removed, the internal mammary artery is eased away from the chest wall by inserting a Leahy gallbladder forceps on the mesial side of the internal mammary artery and vein. In this way the internal mammary artery is carefully displaced laterally. The intercostal vessels are doubly ligated and transected close to the internal mammary artery, with the exception of the sixth intercostal which is ligated and transected approximately ½ inch away from the vessel (Fig. 1, *A* and *B*). The internal mammary vein is then separated from the artery.

(d) *Preservation of periarterial tissue.*—Every attempt is made to preserve perivascular tissue. This is important because we think that branches which develop probably represent a growth of existing vascular channels around the vessel. In a series of animals, we deliberately stripped all tissues surrounding the internal mammary artery, leaving it completely bare. In this group of animals the implantation of a completely bare artery yielded not one single anastomosis.

(e) *Closure of end of artery.*—After the intercostal arteries have been tied between the fourth and sixth, inclusive (sometimes between the third and sixth), the internal mammary artery is then doubly ligated and transected

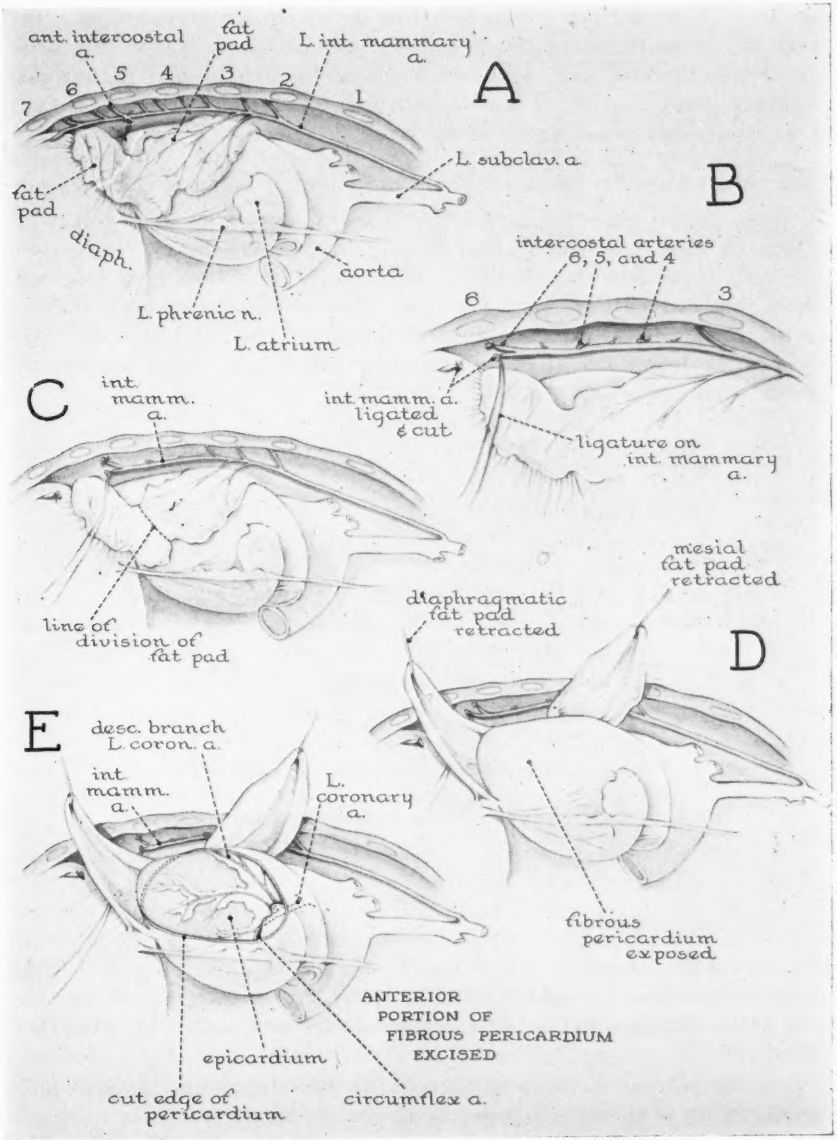


Fig. 1.—Shows technique of preparation of internal mammary artery and pericardial fat pads with excision of anterior fibrous pericardium.

between ligatures well beyond the origin of the sixth intercostal branch. Zero cotton is used, and the ligature on the proximal end of the artery is left long (Fig. 1, *B*). In placing the cotton ligatures, care must be taken to pass them behind the artery without touching it; otherwise, there is danger of injuring that part of the arterial wall which will lie within the myocardium. The distal end of the internal mammary artery is closed, as we believe that there is a better chance of new vessels growing out from the side of the internal mammary artery when there is a strong lateral thrust which occurs as a result of the blood column striking the occluded end of the vessel.

PREPARATION OF PERICARDIAL FAT PADS AND EXCISION OF FIBROUS PERICARDIUM

After the internal mammary artery is prepared, the three pericardial fat pads, each with its separate blood supply, are prepared by dissecting them away from the antero-mediolateral surfaces of the fibrous pericardium, respectively, as shown in Fig. 1, *C* and *D*. The fibrous pericardium is then removed, starting laterally, posterior to the apex, as shown in Fig. 1, *E*. It is necessary to dissect the phrenic nerve and vessels free in order to excise the fibrous pericardium lying beneath them.

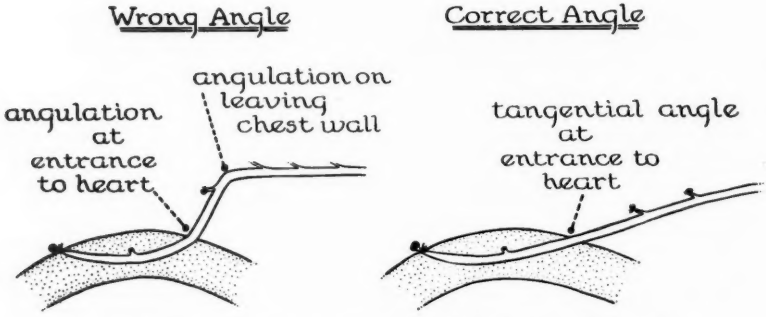

Wrong Angle
Correct Angle
angulation on leaving chest wall
angulation at entrance to heart
tangential angle at entrance to heart

Fig. 2.—Internal mammary artery implantation (anteroposterior plane).

TECHNIQUE OF IMPLANTATION OF INTERNAL MAMMARY ARTERY

(a) *Site of implantation.*—It is necessary to find a portion of the myocardium on the anterior wall of the left ventricle which is not scarred, so that the myocardial tunnel will be made into reasonably healthy muscle. In our experience, this has been possible in every instance. Recently we have had two cases with extensive anterior wall infarctions in which the only healthy myocardium left on the anterior surface of the left ventricle was at the base, close to the division of the left coronary artery, into which the artery was placed.

(b) *Length and direction of tunnel.*—We have found that the most satisfactory length of tunnel is 1 inch. If it is made longer than this, there is a tendency for angulation to occur in the anteroposterior plane (Fig. 2). This will result in occlusion of the artery at its point of entrance into the heart.

The direction of the tunnel is likewise important and must be designed to avoid angulation in the lateral plane (Fig. 3). The artery must enter the heart at a tangent, avoiding acute angles in the lateral or anteroposterior planes.

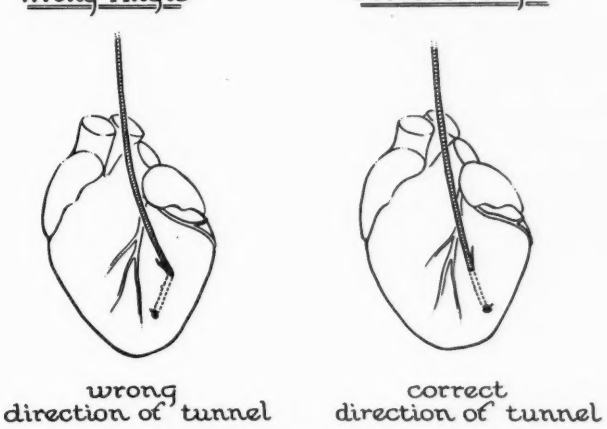


Fig. 3.—Internal mammary artery implantation (lateral plane).

(c) *Method of making tunnel.*—A traction suture of No. 10 cotton is placed at the most distal point of the projected tunnel in the vertical axis of the heart, parallel to coronary vessels. This is tied loosely so as not to occlude any small vessels. Immediately proximal to this traction suture the epicardium is incised with a pointed scalpel (Fig. 4, *F*). A curved mosquito forceps is passed through the opening in the epicardium, and through the myocardial wall for 1 inch. At the point of its emergence, the forceps is spread widely to make sure that the entrance to the tunnel is sufficiently wide so as not to constrict the artery when it is pulled into position (Fig. 4, *G*).

(d) *Depth of tunnel.*—We have found that the greatest number of anastomoses have occurred when the artery is imbedded close to the endocardium. There is, however, danger of opening into the left ventricle, with possible thrombus formation and, for this reason, we place the artery approximately in the middle of the left ventricular wall.

(e) *Entrance to tunnel.*—In making the tunnel by means of a mosquito forceps, it is important to spread the forceps widely before pulling the cotton suture attached to the internal mammary artery through into the tunnel. This stretches the muscle fibers which will surround the implanted artery. In a series of animals we cut these fibers and found resultant scar formation and occlusion at this location. When the fibers are stretched by widening the mosquito forceps, the amount of scar formation at the entrance to the tunnel

appears to be minimal. This is a very important technical point. Too narrow an entrance to the tunnel will occlude the artery immediately. This has been proved by placing a cannula in the end of the artery after it has been pulled into the myocardial tunnel and registering the pressure. When the entrance to the tunnel was too narrow there was no flow through the artery. This was relieved immediately by widening the mouth of the tunnel.

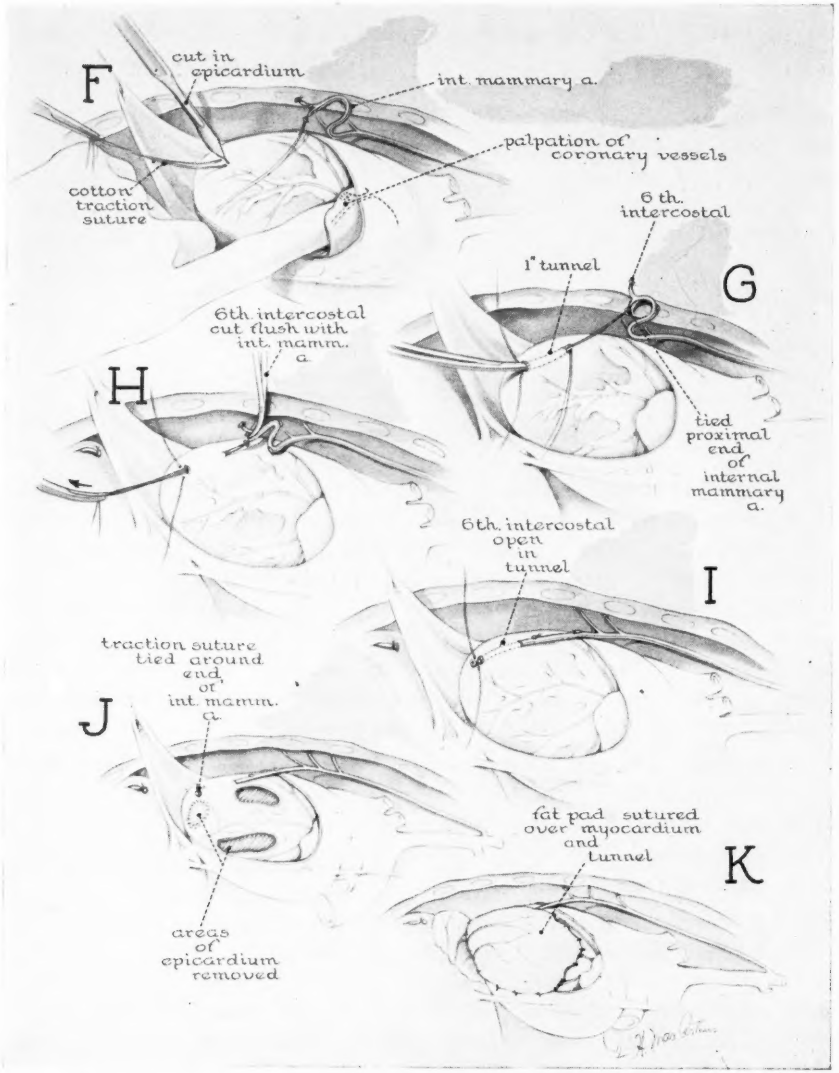


Fig. 4.—Shows the method of making the myocardial tunnel and implantation of internal mammary artery with placement of pericardial fat pads.

(f) *Sixth intercostal artery cut level with internal mammary artery wall.*—Before pulling the artery into the myocardial tunnel, the sixth intercostal artery, which is deliberately left long, is cut with a pair of curved plastic scissors flush with the artery wall. Sometimes the artery is in spasm and does not bleed. It is necessary to be sure that the adventitia around the intercostal is well removed and that an adequate opening can be seen. In early experiments, the sixth intercostal was severed and left long. This was abandoned because we had evidence that the long intercostal artery tended to curl up in the myocardial tunnel and, therefore, might not remain open as frequently as when it was severed close to the main vessel (Fig. 4, *H*).

(g) *Fixation of artery.*—The internal mammary artery is pulled into the tunnel until it projects from the distal end. The fixation cotton suture is then tied around the internal mammary artery, thus securing it in its new position. Very often there will be considerable bleeding from both ends of the tunnel; this bleeding comes from the sixth intercostal vessel, and will eventually stop (Fig. 4, *I*).

(h) *Twist of artery.*—Not infrequently the artery, after it has been severed, twists upon itself and care must be taken in pulling the artery into the tunnel to make certain that it is not twisted. The cut ends of the catgut sutures on the intercostals will indicate quite clearly how the artery should lie.

REMOVAL OF EPICARDIUM AND APPLICATION OF PERICARDIAL FAT PAD GRAFTS DIRECTLY TO MYOCARDIAL MUSCLE

It should be pointed out that in human patients we no longer wrap the internal mammary artery in the mesial pericardial fat pad. Sometimes this pad in man (unlike in animals) is very fibrous and tough and may injure the artery. Therefore, this structure is removed completely, as shown in Fig. 4, *F*. Following the implantation of the internal mammary artery, an attempt is made to remove portions of the epicardium over the anterolateral and posterior surfaces of the heart (Fig. 4, *J*). This usually can be done by means of a fine Allis forceps and scissors. The epicardium usually is removed only between coronary vessels. After this has been done the fat pads are sutured together so as to lie in contact with the bare myocardium (Fig. 4, *K*). When the lung is re-expanded it is necessary to make certain that it does not angle the artery. The chest wound is closed, and the thorax drained.

CONDITION OF THE MYOCARDIUM

There is a vast difference between skeletal and myocardial muscle. The myocardium is composed of a spongelike network of vessels. When the internal mammary artery is placed in the myocardium, it is placed there with an open, bleeding sixth intercostal vessel. The blood from this vessel goes into the tunnel and disappears. A hematoma is never formed. In the heart, due to its spongelike character, blood continues to flow down the internal mammary artery and out through the slit in its wall, thus keeping the artery open until it forms new branches which join with the coronary arterioles

around it. It is our belief that, in making the tunnel, myocardial sinusoids described by Wearn are broken into, and it is into these that the internal mammary artery bleeds (Fig. 5).

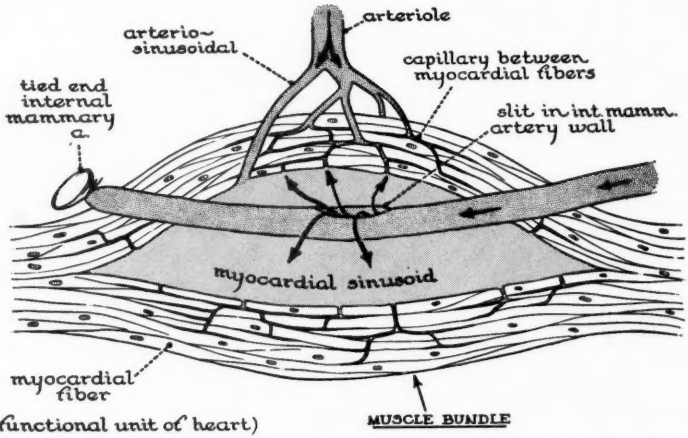


Fig 5.—Mechanism of internal mammary artery branching within the myocardium. Sketch showing myocardial sinusoid lying between myocardial muscle bundles into which the internal mammary artery is placed. The myocardial sinusoidal spaces are in contact with arterioles and capillaries, the latter lying between muscle fibers.

This is a most important point, which is well shown in the following sections. These were made through the site of implant in a human case of angina decubitus operated upon just after the meeting of this Association last year; the patient died from a penicillin reaction eighty-two hours after operation. At the time of operation, certain members of this Association were present and the question was raised as to whether the artery would remain open, as it was not bleeding at the time of implantation. At autopsy, the internal mammary artery was injected with India ink. The ink was seen to flow through the vessels of the left ventricle. Fig. 6, *A* shows a slit in the artery wall with India ink in the space surrounding the vessel. Note that there is no hematoma. Fig. 6, *B* shows India ink in the myocardial arterioles. Fig. 6, *C* shows the ink in large spaces lying between muscle bundle groups. Fig. 6, *D* shows that the India ink has reached the capillaries which lie between muscle fibers. It is logical to assume that, if India ink injected into the implanted internal mammary artery eighty-two hours after operation reached muscle bundles and capillaries, blood must have been flowing along the same pathways. This, we think, is a factor in preventing the artery from becoming blocked.

MYOCARDIAL ISCHEMIA

When myocardial ischemia is present, theoretically it should stimulate the implant to branch and thereby increase blood flow down through the implanted artery, helping to keep it open. Conversely, if there is no need for the implant, the rate of blood flow through it decreases, thereby favoring

occlusion by thrombosis or intimal proliferation. In previous publications we have reported a mammary-coronary anastomosis rate of 71 per cent in ischemic hearts, as compared with 46 per cent in normal hearts (Table I).

TABLE I. FREQUENCY OF MAMMARY-CORONARY ANASTOMOSIS
STANDARD VINEBERG IMPLANT

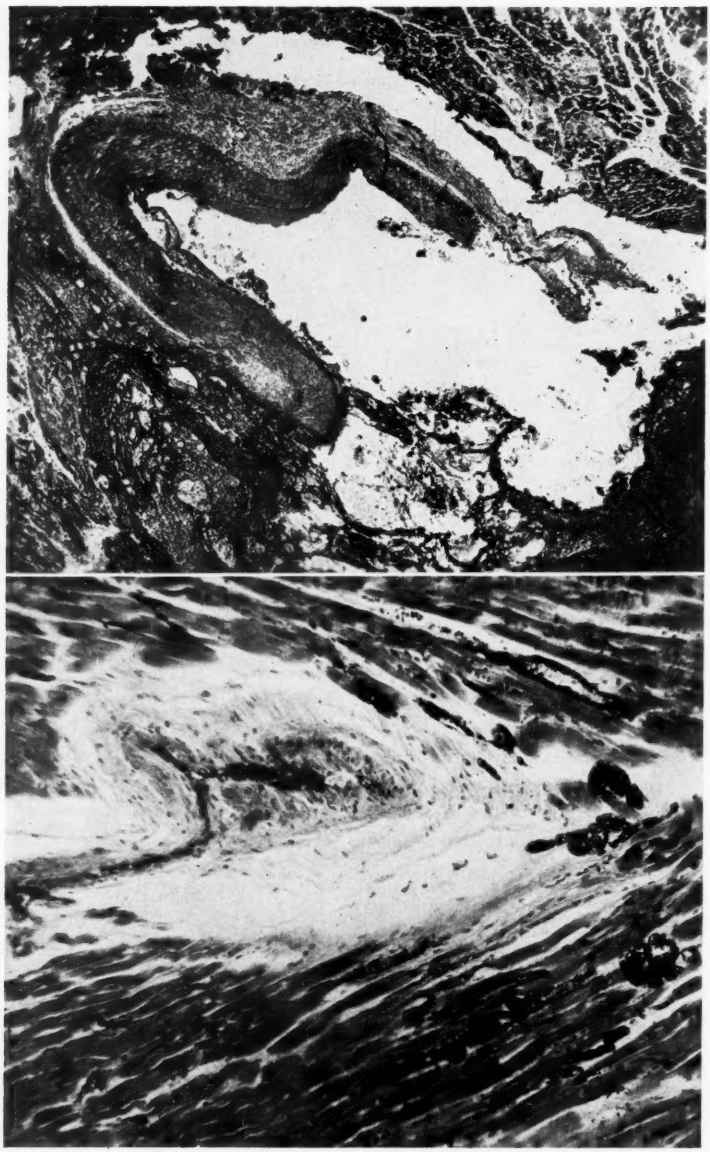
	NO. DOGS	ANTERIOR DESCENDING BRANCH LIGATION	SURVIVED	WITHOUT INFARCT	ARTERY OPEN	ANASTOMOSIS
NORMAL HEART	9 (Vineberg)	Not done	–	–	4+	4
	6 (Niloff)	6	2	1	2	1
	11 (Miller)	11	3	7	7	8
	13 (Buller)	13	5	5	12	5
Total	39	30	10 (30%)	13	25 (65%)	18 (46%)
ISCHEMIC HEART	11 Partial ligation anterior descending branch (23 gauge needle)	8 (72.7%)	5 (45.5%)	7 (63.6%)	6 (54.5%)	
	5 Cellophane wrap anterior descending branch	Exercise tolerance markedly reduced				
	Implant 3 to 4 months later	Improved		4 (80%)	4 (80%)	
	5 Cellophane wrap anterior descending branch plus internal mammary artery implant	Exercise tolerance reduced in 2		2 (100%)	2 (40%) Small	
		Exercise tolerance maintained at preoperative levels in 3		3 (100%)	3 (60%) Large	
Total	21			16 (76%)	15 (71%)	

In this table, figures are shown to indicate how 65 per cent of implanted internal mammary arteries remained open when placed in normal hearts, and formed anastomoses in 46 per cent. When the artery was placed in ischemic hearts, the vessels remained open in 76 per cent, and formed mammary coronary anastomoses in 71 per cent.

Now let us examine serial histologic sections of internal mammary arteries placed in normal hearts. Here we find, as in all implants, that the portion of the vessel distal to its open branches has a greatly reduced lumen as compared to the same vessel proximal to open branches.

Serial sections shown in Fig. 7, *A* and *B* were taken through a 4-month-old internal mammary implant in a normal heart. The section in Fig. 7, *A* was taken proximal to the open branches. There is some intimal proliferation. However, the actual lumen is still 70 per cent of the potential lumen and it measures 1.2 by 0.5 mm. This is a good-sized vessel, capable of delivering a large quantity of blood to the myocardium. Fig. 7, *B* shows the same vessel distal to the open branches. The lumen has been reduced to 10 per cent of its potential size. These sections indicate that an artery left in a normal heart shows intimal proliferation throughout. The extent of the proliferation varies, depending on whether the section is taken proximal or distal to open branches.

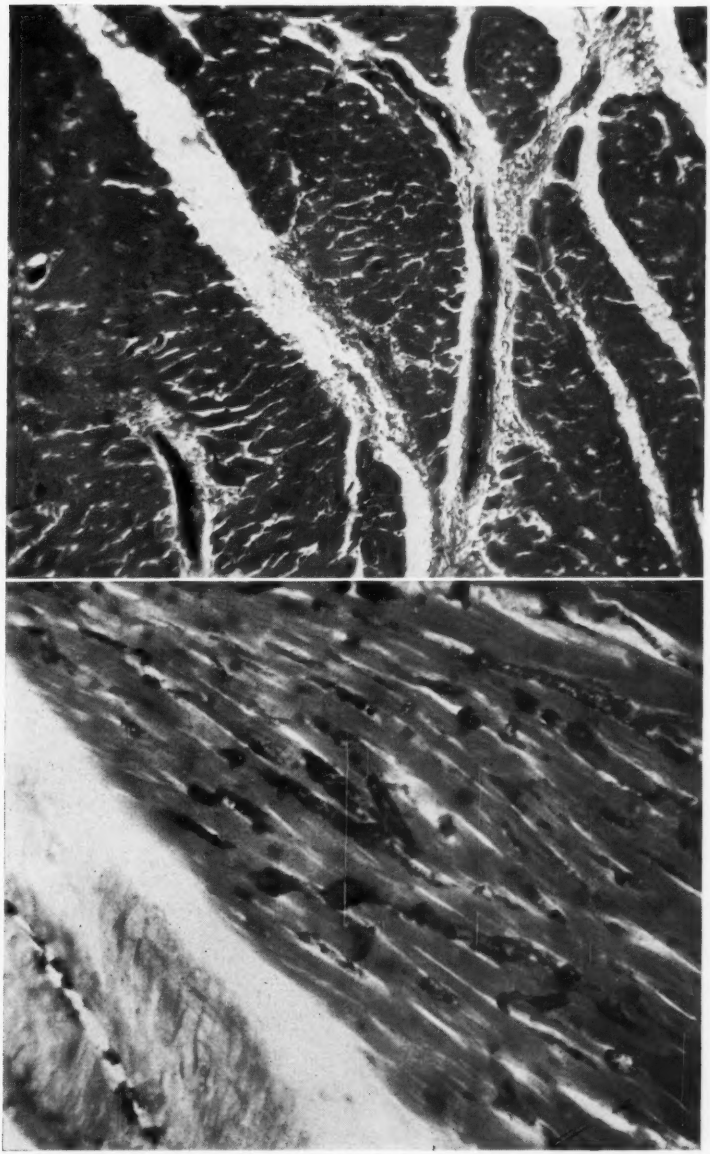
A.



B.

Fig. 6, *A* and *B*.—Internal mammary artery in myocardium of human case of angina decubitus. This patient died eighty-two hours after implantation from intractable bronchial spasm following a penicillin injection. At autopsy, the internal mammary artery was injected with India ink. *A*, Note the slit in the arterial wall, with India ink in the space surrounding the vessel. There is no hematoma. *B*, Note India ink filling the arterioles.

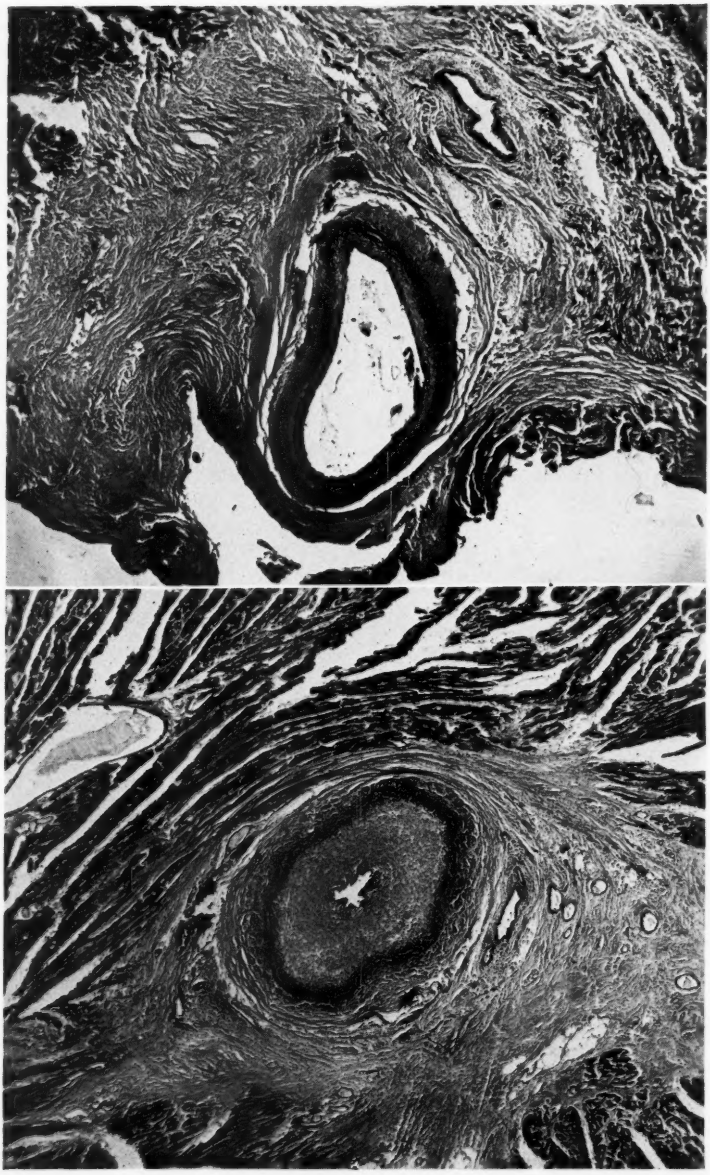
C.



D.

Fig. 6, *C* and *D*.—Same patient as in Fig. 6, *A* and *B*. *C*, Note how the India ink is lying in large spaces between muscle bundle groups. These may be myocardial sinusoids filled with India ink. *D*, Note how the India ink injected through the internal mammary artery has reached the capillaries which lie between muscle fibers.

A.



B.

Fig. 7.—Serial sections taken through site of implantation of an internal mammary artery placed in a normal heart four months previously. *A*, Serial section proximal to open branches lying within the myocardium. There is some intimal proliferation. Actual lumen is 70 per cent of potential, and measures 1.2 by 0.5 mm. *B*, Serial section of same vessel, distal to open branches. Lumen has been reduced to 10 per cent of its potential size.

The sections in Fig. 8, *A* and *B* were taken from a 58-week-old implant left in a normal heart. Fig. 8, *A* shows a section taken just proximal to the branching of the internal mammary artery. In this 58-week-old implant left in a normal heart, intimal proliferation has developed proximal to the open branches, resulting in a markedly reduced lumen which amounted to 20 per cent of its potential. The actual measurements of this lumen were 0.5 by 0.42 mm. In Fig. 8, *B*, taken through the artery beyond the open branches in the same animal, the lumen is completely occluded. In these animals no attempt was made to promote myocardial ischemia. The implanted internal mammary arteries were in competition with normal coronary vessels. The pressure differential between the implanted internal mammary artery and the surrounding myocardial vessels was not great enough to maintain a rapid flow of blood through the implanted artery. The resultant intimal proliferation gradually diminished the size of or completely occluded the lumen. This process is very similar to that which occurs in other unused vessels in the human body, such as the ductus arteriosus, etc.

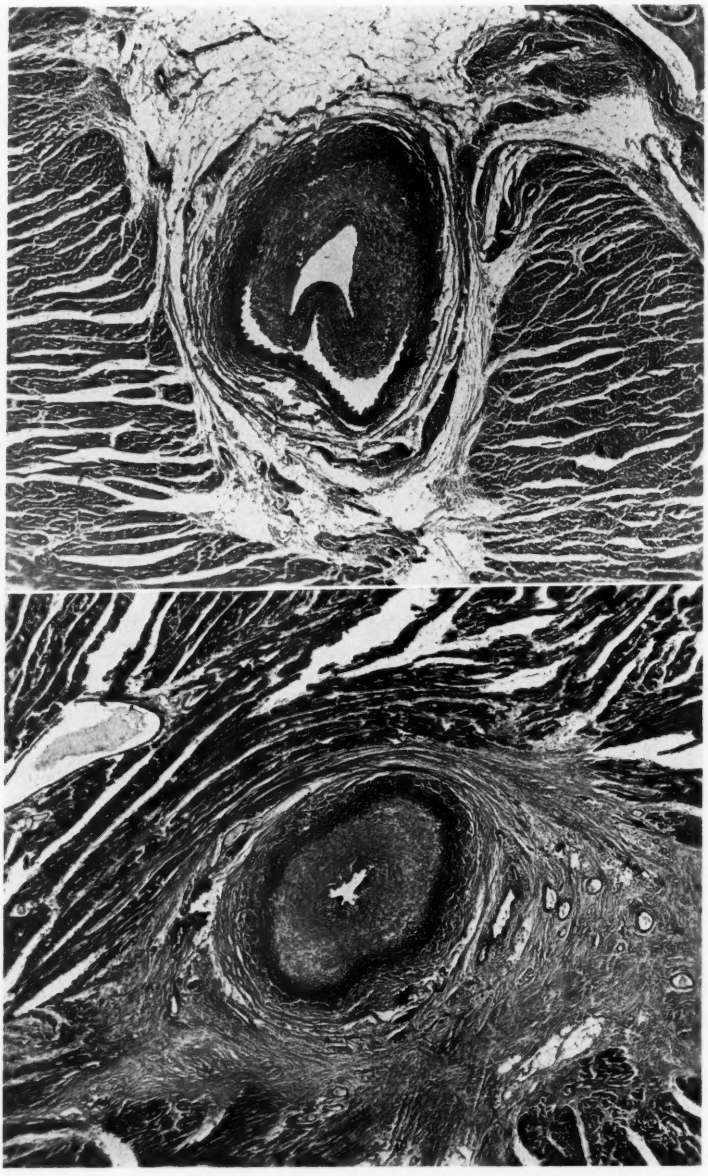
ISCHEMIC HEART

Examination of serial sections taken through the internal mammary artery placed in an ischemic heart shows that the artery proximal to open branches is practically free of intimal proliferation. The intimal proliferation, however, is definitely present distal to open branches, as is demonstrated by the sections shown in Fig. 9, which were taken four months after implantation and three months after ligation of the anterior descending branch.

The section in Fig. 9, *A*, taken just proximal to branching, shows no intimal proliferation of the implanted vessel. Fig. 9, *B* shows the implanted artery distal to the branches. Note how the artery in this location has been almost completely occluded.

The size of the implanted internal mammary artery has been outlined by digestion casts in seven animals in which the internal mammary artery was forced to carry the load six weeks after implantation due to ligation of the anterior descending branch at this time. These arteries were then studied five, six, and seven months after implantation. Fig. 10 outlines the size of the lumen of such an artery along with its branches, seven months after implantation. It is clearly as large as a major coronary vessel. The amount of blood carried by this artery to an ischemic heart has been found to be 27.6 to 29 c.c. per minute. It is clear, assuming that the artery has not been damaged or angled, that the frequency and duration of anastomosis is related to the conditions of the soil into which the artery is implanted. In human cases of coronary artery insufficiency the soil, theoretically, should always be favorable when the artery is placed in muscle and away from a scar. It is interesting to note that of 9 human patients who have died following internal mammary artery implantation, 7 died ten hours to ten days after operation; 6 had open arteries. That is a rate of 85.7 per cent, which is approximately the percentage of coronary anastomoses that have occurred in animals. The one artery which was thrombosed was badly angled due to a marked elevation of the left diaphragm caused by paralytic ileus.

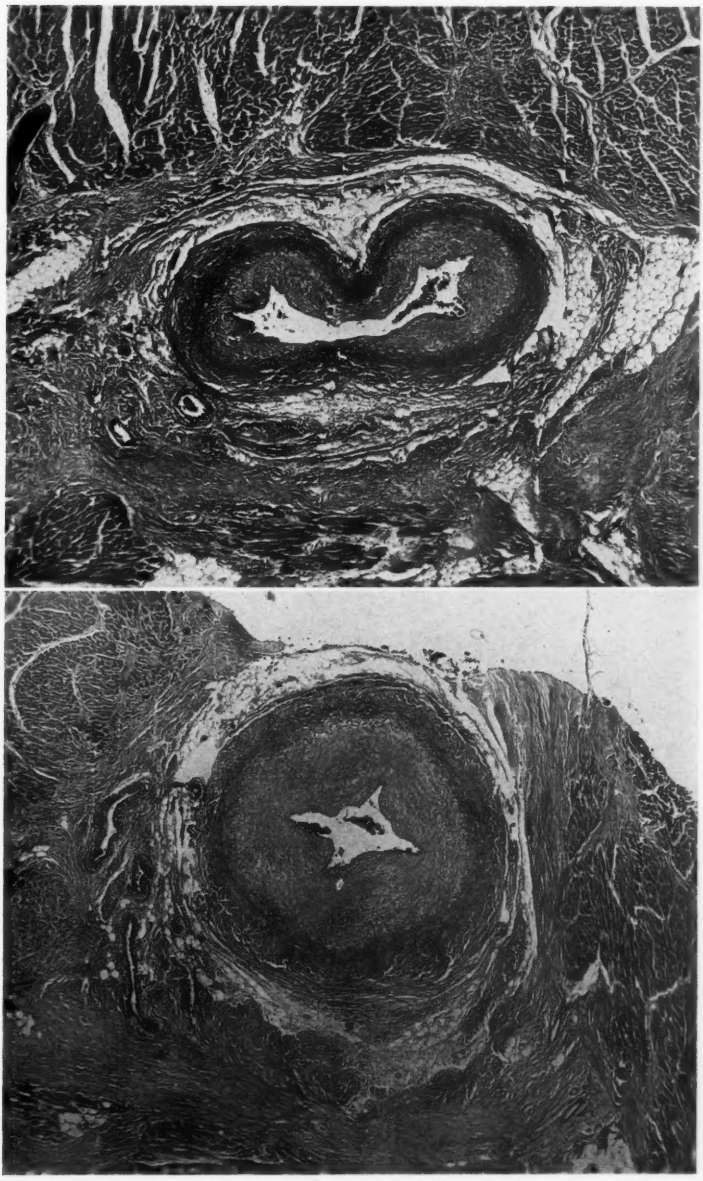
A.



B.

Fig. 8.—Serial sections taken through site of internal mammary artery implant placed in a normal heart for fifty-eight weeks. *A*, Serial section proximal to open branches. Note how intimal proliferation has greatly reduced the lumen of the vessel. *B*, Serial section of the same vessel distal to open branches, showing the lumen practically obliterated.

A.



B.

Fig. 9.—Serial section studies of internal mammary artery placed in ischemic heart in animal No. 162. Sections were taken four months after implantation and three months after ligation of anterior descending branch. *A*, Serial section taken just proximal to branching of the artery within the myocardium. There is no, or little, intimal proliferation at the end of four months. *B*, Same vessel distal to branches which join up with coronary vessels. No marked proliferation, with decrease in size of lumen.

At present, as in the past, we are striving to increase the mammary-coronary anastomosis rate.

At the suggestion of Sir James Learmonth, we have transected the left subclavian artery distal to the internal mammary artery. It is implanted into the left ventricle with many slits in its sides, from which blood pours into the myocardial tunnel. The value of this modification of the implant procedure is not yet established and, therefore, has not been used in the treatment of human cases of coronary artery disease.

Fig. 10.—Digestion cast made by injecting the implanted internal mammary artery seven months after implantation and five months after ligation of anterior descending branch. Flow rates through this artery into the arterioles of the ventricle were 27.6 to 29 c.c. per minute.

RESULTS

Mortality.—A total of 45 patients suffering from coronary artery insufficiency have been treated by internal mammary artery implantation. In the 33 patients who had no angina at rest, there were 2 deaths, a mortality rate of 6.6 per cent. In the 12 patients suffering from angina decubitus, there have been 7 deaths, a mortality rate of 58.3 per cent. The mortality rate of the entire group was 20 per cent (Table II).

TABLE II. SUMMARY OF MORTALITY RATE IN 45 PATIENTS WITH CORONARY ARTERY INSUFFICIENCY TREATED BY INTERNAL MAMMARY ARTERY IMPLANTATION

	NO. OF PATIENTS	DEATHS	MORTALITY RATE (PER CENT)
No angina at rest	33	2	6.6
Angina decubitus	12	7	58.3
Entire group	45	9	20

Deaths Occurring Before Operation (Table III).—It is interesting to compare the operative mortality with the mortality rate occurring before operation. We have had 3 patients die in the hospital and 4 outside the hospital while awaiting operation. A total of 7 patients died awaiting operation; there was a total of 9 operative deaths. Almost as many patients have died awaiting operation as have died postoperatively.

TABLE III. DEATHS OCCURRING BEFORE OPERATION

Three patients have died in hospital from acute coronary occlusion and infarction while awaiting operation	
Four patients have died suddenly outside hospital while awaiting admission for operation	
Total deaths awaiting operation	7
Total operative deaths out of 45 cases	9
Almost as many patients have died awaiting operation as have died postoperatively	

PREOPERATIVE CONDITION OF FORTY-FIVE PATIENTS WITH CORONARY ARTERY INSUFFICIENCY TREATED BY INTERNAL MAMMARY ARTERY IMPLANTATION

Results.—An analysis of the condition of 45 patients prior to operation has been made. Table IV shows that 78 per cent were not working and that the other 22 per cent were working and using nitroglycerine. Fifty-six per cent could walk no more than 200 feet, and 44 per cent showed an average of seventeen steps on the two-step test. Fifty-three per cent of the patients showed dyspnea and 53 per cent showed marked fatigue. Eighty-four per cent had had one or more infarctions.

TABLE IV. CONDITION OF 45 PATIENTS WITH CORONARY ARTERY INSUFFICIENCY TREATED BY INTERNAL MAMMARY ARTERY IMPLANT

Not working		34	(78%)
1 to 4 years	22		
1 to 11 months	12		
Working (with nitroglycerine) 1 to 300 tablets weekly		11	(22%)
Exercise tolerance:			
0 to 200 feet		25	(56%)
Two-step (average, 17 steps)		20	(44%)
Dyspnea		24	(53%)
Orthopnea		2	
Marked fatigue		24	(53%)
One or more previous infarctions proved		38	(84%)

Forty Cases of Human Coronary Artery Disease Followed Six Months to Four and One-half Years.—The results of 40 human cases with advanced coronary artery insufficiency, treated by internal mammary artery implantation and followed from six months to four and one-half years since operation, are shown in Table V.

There were 30 patients who had no angina at rest. Of this number, 23 (77 per cent) were totally disabled prior to operation. Twenty-eight survived the operation, and 16 (57.2 per cent) had no pain or slight pain. In 4 (14.2 per cent) the pain was less. Those with no pain or slight pain totaled 20 (71.4 per cent of the group). There were 7 patients who were the same or worse. Twenty-three (77 per cent) of the group returned to work after operation. This is a complete reversal of their condition before operation.

TABLE V. RESULTS OF CORONARY ARTERY INSUFFICIENCY TREATED BY INTERNAL MAMMARY ARTERY IMPLANTATION IN 40 PATIENTS FOLLOWED SIX MONTHS TO FOUR AND ONE-HALF YEARS

	BEFORE OPERATION			AFTER OPERATION			
	NO. OF PATIENTS	TOTALLY DISABLED	SURVIVED	NO PAIN OR SLIGHT PAIN	LESS PAIN	SAME OR WORSE	RETURNED TO WORK
No angina at rest	30	23 (77%)	28 (93.3%)	16 (57.2%)	4 (14.2%)	7 (25%)	23 (77%)
					71.4%		
Angina decubitus	10	10 (100%)	5	1	3	1	3

Four patients with no angina at rest have died months to years after operation

Twenty-four (85.7%) of this group are still alive. Eighteen (64.2%) have no pain or less pain. Nineteen (67.8%) still work

No change in the angina decubitus group

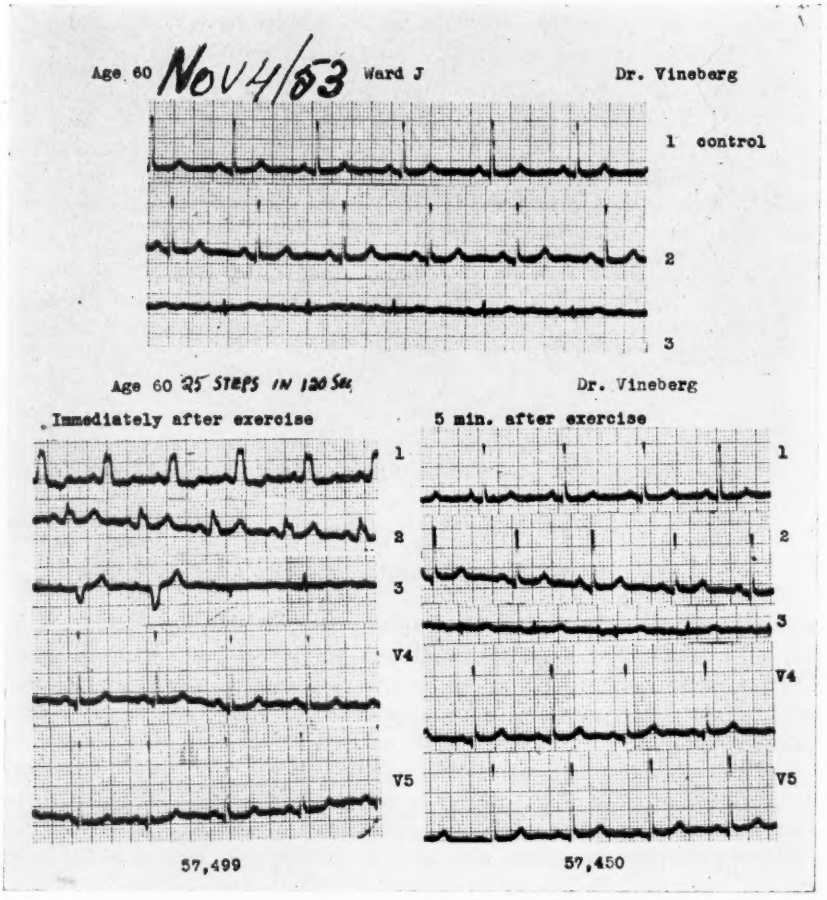


Fig. 11A.—Electrocardiogram of a 60-year-old woman whose exercise tolerance on Nov. 4, 1953, was twenty-five steps in 120 seconds, before the development of pain, dyspnea, and left ventricular bundle branch block.

Your attention is drawn again to the fact that 23 (77 per cent) were totally disabled prior to operation, and 23 (77 per cent) returned to work after operation.

There have been 4 late deaths in the "no angina at rest" group, months to years after operation. The causes are listed in Table VI.

Twenty-four patients (85.7 per cent) of the group are still alive, and 18 (64.2 per cent) have no pain, or less pain, while 19 (67.8 per cent) still work.

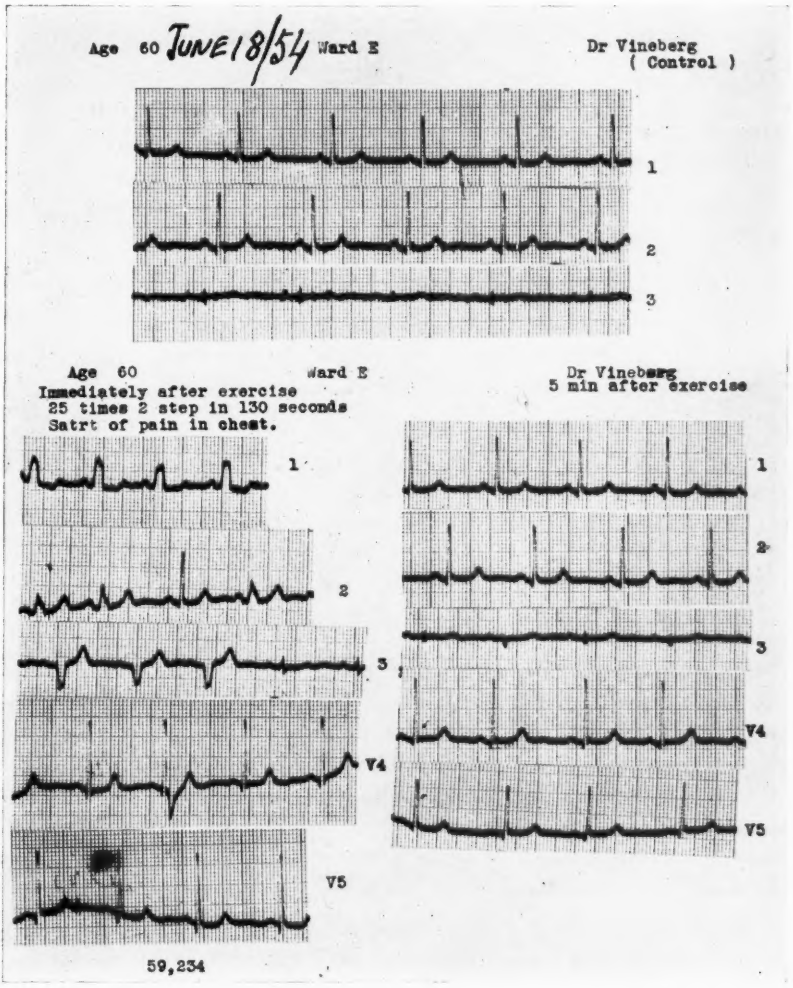


Fig. 11*B*.—Electrocardiogram of same patient as in Fig. 11*A*, whose exercise tolerance on June 18, 1954, was twenty-five steps in 130 seconds before development of pain, dyspnea, and left ventricular bundle branch block.

TABLE VI. CAUSES OF LATE DEATHS FOLLOWING INTERNAL MAMMARY ARTERY IMPLANTATION IN 45 CASES OF CORONARY ARTERY INSUFFICIENCY

One case (third patient) had steady increase in pain; Died four years postoperatively of aplastic anemia (artery had pulled out of heart)
One case, totally incapacitated preoperatively, worked one and one-half years; died from rupture of old heart scar while pushing car out of snow
One case, totally incapacitated preoperatively, ran a cattle ranch for two years; died three years postoperatively after riding horseback
One case, error in diagnosis. Large anterior scar, right ventricle; left ventricle normal; artery placed in left ventricle; no improvement; steadily worse; died six months postoperatively

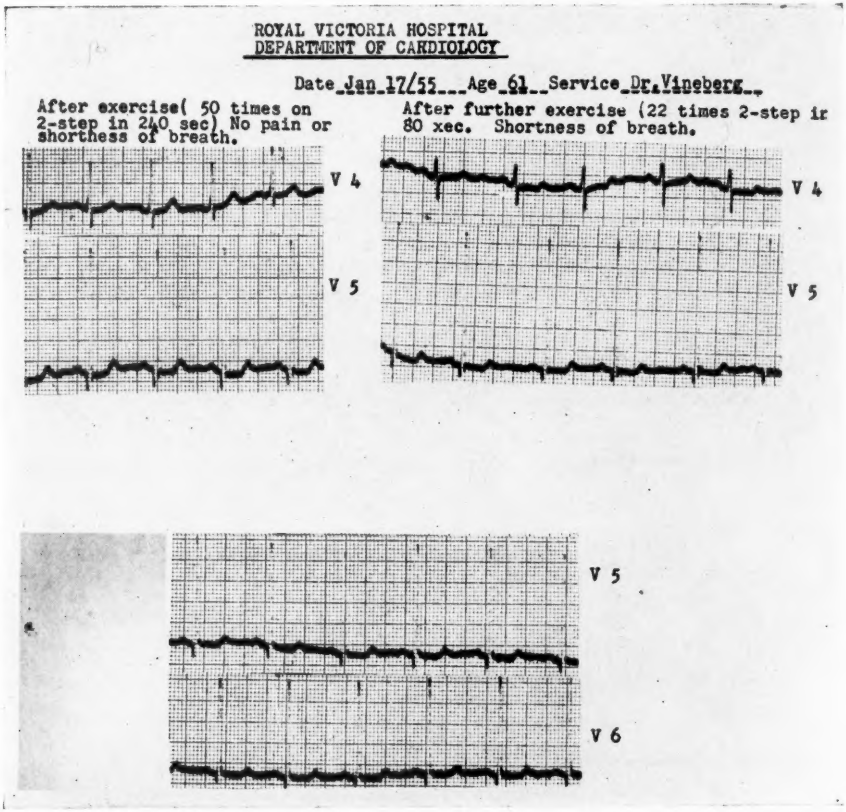

ROYAL VICTORIA HOSPITAL
DEPARTMENT OF CARDIOLOGY
Date Jan 17/55 Age 61 Service Dr. Vineberg
After exercise(50 times on 2-step in 240 sec) No pain or shortness of breath.
After further exercise (22 times 2-step in 80 sec. Shortness of breath.
V 4
V 5
V 4
V 5
V 5
V 6

Fig. 12.—Electrocardiogram of patient in Fig. 11, six months after internal mammary artery implantation. Patient is free of pain and dyspnea. Note tremendous increase in exercise tolerance without the development of pain or left ventricular bundle branch block.

In the angina decubitus group there were 12 patients, all totally disabled. Five survived operation, 4 are improved, and 3 have returned to work.

Our results in the treatment of coronary artery insufficiency by internal mammary artery implant are, as you can see, most encouraging. Before concluding, we should like to present a rather typical case.

Mrs. M. G., aged 60 years, was first admitted to the Royal Victoria Hospital because of severe angina in November, 1953. Her exercise tolerance was limited to twenty-five steps in 120 seconds before she developed pain and dyspnea. Electrocardiograms taken at the height of the pain showed left ventricular bundle branch block. There had been a history of peripheral edema and, because of the dyspnea, our medical group preferred not to subject her to operation. In June, 1954, this patient had to stop working because of her inability to walk one-half block. She was readmitted to the hospital where a second exercise tolerance test showed, once again, a limit of twenty-five steps before the onset of pain and dyspnea. Again, the left ventricular bundle branch block was shown on the ECG at the height of pain (Fig. 11). In July, 1954, she received an internal mammary artery implant, and returned to work in September, 1954, free of pain and with a markedly increased exercise tolerance. The dyspnea had disappeared.

On Jan. 17, 1955, this patient was re-exercised and after fifty steps in 240 seconds there were no pain, no dyspnea, no increase in heart rate, and—more important—no evidence of left ventricular bundle branch block! Because the pulse rate had not gone up, she was immediately re-exercised at a faster pace. After twenty-two steps in eighty seconds, she developed slight dyspnea. There were, however, no pain and no evidence of left ventricular bundle branch block (Fig. 12).

It is our opinion that there is some objective evidence of improvement in this case!

SUMMARY

The technique of internal mammary artery implant is given and some of the factors which favor mammary-coronary anastomosis are discussed.

Evidence has been presented to show that the frequency of mammary-coronary anastomosis is dependent upon (1) technique of the preparation of the artery, (2) technique of implantation, and (3) the condition of the myocardium into which it is implanted. The duration of the anastomosis is dependent upon the need for it. When the internal mammary artery is placed in ischemic animal hearts, there is a mammary-coronary anastomosis rate of 71 per cent. In 7 human cases examined ten hours to ten days after implant, the artery was found to be open in 71 per cent.

Intimal proliferation occurs to a slight extent in all implanted internal mammary arteries. This, however, increases when the blood flow through the artery is slowed. Thus, in the same implant, intimal proliferation may be absent proximal to open branches and very marked distal to them.

When an artery is placed in an ischemic heart, there is practically no intimal proliferation proximal to open branches.

The mortality rate of 45 patients treated by implant is given, and the results in 40 patients followed six months to four and one-half years are presented. The results in the treatment of human cases of coronary heart disease are very satisfactory. Seventy-eight per cent of the group were not working prior to surgery, and 77 per cent have returned to work.

The patients referred to have been operated upon at the Royal Victoria Hospital, Jewish General Hospital, Queen Mary Veterans' Hospital, and the Institute of Cardiology, all of Montreal.

We wish to express our appreciation for the assistance of Drs. G. Lyman Duff and G. C. McMillan, of the Department of Pathology, Royal Victoria Hospital, and Dr. Rene Mailloux, of the Institute of Cardiology, Montreal.

Discussion

(Papers by Leighninger; and Vineberg and Buller)

DR. FREDERICK R. MAUTZ, Cleveland, Ohio.—The crucial proof of the success of a procedure designed to increase collateral blood flow to an ischemic segment of myocardium would seem to be the actual demonstration and measurement of increased blood into the area under physiologic conditions approaching the normal. In 1941, Beck and associates, in carefully controlled experiments, showed that abrasion of the heart one to three weeks prior to descending ramus coronary artery ligation reduced the mortality from a well-established control value of 70 per cent to 38 per cent, and appeared to reduce the degree of infarction in the survivors. Critics raised the question as to whether factors other than improved circulation might be responsible for these results.

In 1937, Gregg and I postulated that the events taking place within an occluded coronary artery should give the best indication of the development of collateral circulation and also provide a means of determining the exact sources of the blood. At that time we were able to demonstrate the spontaneous development of collateral circulation following occlusion of coronary arteries, both by acute ligation and by progressive partial occlusion in stages. It was thought that this would serve as a base line for the study of procedures designed to augment the spontaneous development of collateral. The amount of collateral that developed varied greatly from little or none in some instances to what might be termed a massive collateral in other instances. The amount of collateral flow seemed to vary inversely with the degree of infarction. In these instances the primary stimulus to the development of collateral appeared to be arterial obstruction with resultant hypoxia of an area of myocardium. No effort was made to augment the development of the collateral, although the effect of surgical exposure of the heart for the purpose of occluding the coronary arteries remained as a factor to be evaluated.

In trying to establish the source of the collateral, two-thirds or more could be definitely shown to come from the unoccluded coronary arteries. We did not succeed in tracing the source of one-tenth to one-third of the collateral, except that the quality of the blood was indistinguishable from arterial blood on the basis of oxygen and carbon dioxide content. Some of it, no doubt, came from the septal artery and the remainder from basal extracoronary anastomoses. These findings were in agreement with the anatomic observations presented to this Association in 1937, namely, that by far the greatest increase in size of collateral channels was in the intercoronary anastomoses.

The importance of Dr. Leighninger's work rests upon the fact that he has shown, by retrograde blood flow studies, that factors other than coronary arterial obstruction will significantly increase basic arterial collateral flow, namely, abrasion of the epicardium, chemical irritation of the epicardium, and partial coronary sinus obstructon. This work beautifully complements the tests of benefit previously used by Beck and associates, namely, (1) reduction in mortality and (2) size of infarct. Dr. Leighninger's work clearly invalidates any hypothesis that the beneficial effects of these procedures are fortuitous or are the result of factors other than improved collateral circulation.

We can now say without reservation that the studies presented by Dr. Leighninger provide the crucial proof, which our medical colleagues have so long demanded, that collateral arterial flow to the myocardium can be increased by surgical methods in terms of actually measured blood flow under normal circulatory conditions.

DR. ALFRED GOLDMAN, Beverly Hills, Calif.—(Slide) I wish to show another method for augmenting coronary circulation which we have been studying experimentally in dogs. This method involves the transplantation of a U-shaped arterial graft obliquely through the myocardium so that the multiple openings in the graft simulate the thebesian-sinusoidal-luminal system and coronary blood from the left ventricular cavity to its myocardium.

(Slide) This is a method of evaluating the size of the infarcts when the dogs are challenged after being prepared two months previously with the grafts. Challenge consists of ligation of the anterior descending coronary artery.

(Slide) This is the method developed by Dr. Sherman Strauss for measuring the two diameters of the infarcts. Here are the sections from which the diameters are measured. The slices at right angles to the anterior descending coronary artery are 2 to 3 mm. thick. The third dimension, or N-number, is estimated from the character and extent of the infarcted myocardial thickness. When the diameters of the infarcted area are compared with the diameters of the heart itself and infarct ratio is computed and the IR index is multiplied by the N-number, a numerical value for the infarct size is obtained. In our present preliminary stage the infarct size is represented by the numerical value and has not been found to be significantly decreased.

(Slide) Here is the microphotograph of the graft in situ showing the lumen of the blood vessels parallel with the graft.

(Movie) A colored motion picture, showing another method of electrocardiographic exploration of the human ischemic heart, was shown. Dr. Myron Prinzmetal has been in charge of the electrocardiographic interpretation.

DR. J. H. WALKER, Charleston, W. Va.—I would like to record my experience with the Vineberg procedure. Twenty-four patients have been operated upon for coronary insufficiency following myocardial infarction. There has been one fatality, and this was prior to placing the artery in the myocardium. There are 23 living patients. One patient still requires nitroglycerine but in much smaller quantities; three patients are too recent for follow-up; one has minimal congestive failure; 18 have had good results. In some of the 23 living patients, I have modified the procedure somewhat in that talcum powder poudrage has been used, as advocated by Dr. Thompson, along with the internal mammary artery transplant. I cannot see any definite difference between the procedure as done primarily by Dr. Vineberg or with the addition of poudrage.

DR. J. D. FRYFOGLE, Detroit, Mich.—I stand in awe and appreciation of the wonderful work of Drs. Beck and Leighninger. This stimulated me, in 1952, to perform my first Beck II procedure. The patient did so well, after being a cardiac invalid, that he now makes all our cardiac instruments. There is no question, I think, that, at least for the first ninety days, the formation of intercoronary channels resulting from a free vein graft or direct arterialization of the coronary sinuses does occur. Speaking in terms of thrombosis, which has plagued Drs. Vineberg and Beck for awhile with the vein graft, and thinking also of the patient's trepidation about a second operation after arterialization, I sought in our own little laboratory to arrive at a means whereby we could accomplish the same end with a single operative procedure. I would like to show the technique of this.

(Slide) This will orient you with the typical Beck procedure, with the free vein graft coming between the aorta and the coronary sinus.

(Slide) You do the first part of this operation (here is the coronary sinus and here is the free vein graft). Then you take the small, gunlike instrument as pictured. To it is attached a siliconized polythene plastic cuff. The idea of obtaining arterial blood from the ventricular cavity originated with Dr. Thomas O'Neill of Philadelphia. This piece of polythene plastic is excellent for the procedure; it fits directly over the sleeve of the punch and it has an imbedded silk suture in its neck. There are two little special needles curved on the end. You advance the instrument through a sleeve as demonstrated.

(Slide) There is a simple purse-string suture around the sleeve which controls bleeding. You push the instrument through the ventricular wall and are able to punch out, with a single motion, a nice round hole. The secondary sleeve pushes the plastic prosthesis into the cardiac wall. This has not been done on any human patients, but it has been done

on 23 dogs. The coronary sinus is never in the same place; sometimes it is up in the auricle wall, sometimes down in the ventricle wall, and sometimes right over the valve itself. By placing the finger through the auricular appendage, you can direct exactly where in the heart wall you want to place the prosthesis.

(Slide) Then you place the plastic prosthesis where you want it, sew it into the myocardium, and tie outside the wall. You are now ready to close the graft sleeve and cut off the excess.

This is an easy operation to do and it accomplishes a free flow of blood from the left ventricular cavity. It allows two things that I think are important in preventing thrombosis. First, you do not distort, in any way, the coronary sinus. Second, you are able to tie down the sinus to 3 mm. at the time of operation and do not get rupture of the little venules over the heart wall, as you do when you are obstructing aortic pressure. In the aorta you have systolic pressure and diastolic pressure, and I think that the constant diastolic pressure is the factor that makes the small venules rupture. When you have direct communication to the left ventricular cavity you have a systolic pressure, true, which permeates the heart wall, but a zero diastolic pressure, thus allowing the vein to rest. This, I think, is the reason we do not get rupture of the small peripheral veins. Six of the 23 dogs have been kept for duration studies. These will be reported in subsequent writings and I ask that this be accepted as a preliminary report.

DR. J. L. EHRENHAFT, Iowa City, Iowa.—Ever since seeing Dr. Vineberg's work in Montreal last year, we have been very much impressed by the possibility of creating a third coronary artery by vessel implantation into the myocardium. Recently we have used the splenic artery in dogs for myocardial implantation. This vessel is quite long and can easily be brought up from the abdomen through the diaphagm. Obviously, this is only an experimental procedure that is possible in dogs because the spleen and splenic arteries in this animal are very easily mobilized. I doubt very much that this vessel could be used in the human patient. We have found that those vessels, after implantation, seem to remain patent and probably form anastomoses with the other coronary vessels.

DR. DAVID S. LEIGHNINGER, Cleveland, Ohio (Closing).—I would like only to thank the discussors.

DR. VINEBERG (Closing).—I wish to take this opportunity to congratulate Dr. Leighninger, whose work I have been following with much interest for a number of years.

I have had no definite experience with the Mautz and Gregg method of studying intercoronary arterial communication. In our laboratory, many types of revascularization procedures have been tried, which include attempts at making artificial thebesian canals, cardio-omentopexies, pericardial fat grafts, coronary vein ligation, and talc and implant procedures. In 55 animals, where there has been no evidence of revascularization, animals died in 83.1 per cent of the cases when the anterior descending branch of the left coronary artery was ligated. In 40 animals, in which there was revascularization due to adequate mammary-coronary anastomoses, there were no deaths.

In the first group of 55 animals, in which various types of operations designed to promote revascularization failed to do so, the operative interference in itself did not protect these animals against anterior descending branch ligation.

Because of this information, I cannot agree with Dr. Beck and his associates, who have indicated that, no matter what is done to a heart, the procedure in itself promotes the development of collaterals. This statement may be true in part, but certainly, in our experience with animals, these collaterals were not functioning six weeks to two months after unsuccessful attempts at revascularization in normal animals' hearts by different types of revascularization procedures.

The coronary arteriolar tree is divided into three separate and distinct zones (Fig. 1). These zones in the normal heart usually have no communication. When coronary artery disease intervenes, or ischemia of the heart is promoted through other means, communications between these separate arteriolar zones develop and, in this way, collateral vessels are formed (Fig. 2). It seems to us that this is the only time that the term "collateral" should be used in reference to coronary artery disease. Since coronary disease

usually involves the epicardial portions of the major vessels, it is very difficult to conceive how the development of collaterals between three separate arteriolar zones can bring fresh blood past the blockage which occurs in the proximal portion of the major coronary arteries.

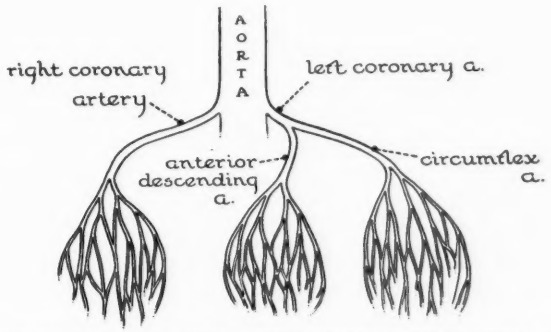


Fig. 1.—Arteriolar zones (normal heart). Three distinct arteriolar zones; normally no connections between zones; no collaterals.

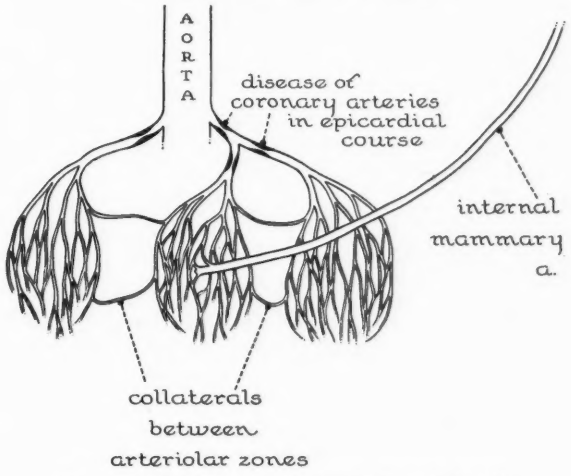


Fig. 2.—Arteriolar zones (ischemic diseased heart).

The methods which aim at stimulating the development of intercoronary or collateral circulation, such as have been described by Beck and associates, in our opinion are bound to fail, because what they attempt to do has already been done by Nature. It has been shown by many pathologists, including Schlesinger, Zoll, and Blumgart, that 98 per cent of human cases of coronary artery insufficiency develop extensive collateral pathways between the various arteriolar zones. Therefore, operations on human cases of coronary artery insufficiency that are designed to promote collateral circulation would appear to be unnecessary, in so far as that which they are supposed to do has already been done. What is necessary in coronary artery insufficiency is a powerful new source of extracardiac blood which is brought to the ischemic myocardium in such a manner as to bypass the occluded proximal portions of the major coronary vessels.*

*References: Schlesinger, M. J.: An Injection Plus Dissection Study of Coronary Artery Occlusions and Anastomosis, Am. Heart J. **15**: 528-568, 1938; Schlesinger, M. J.: Relation of Anatomic Pattern to Pathologic Conditions of Coronary Arteries, Arch. Path. **30**: 403-415, 1940; Zoll, P. M.: Normal and Pathological Anatomy of the Coronaries, Tr. Am. Coll. Cardiology **1**: 29, 1951.

AN ANALYSIS OF FIFTY PATIENTS TREATED BY MITRAL COMMISSUROTOMY FIVE OR MORE YEARS AGO

ROBERT P. GLOVER, M.D., THOMAS J. E. O'NEILL, M.D., AND
O. HENRY JANTON, M.D. (BY INVITATION)
PHILADELPHIA, PA.

INTRACARDIAC surgery for certain types of congenital and acquired diseases is now a well-established and highly successful adjunctive and definitive form of treatment. The mitral valve, the commonest site of intracardiac pathology, has naturally occupied the center of attention during these developmental years, for the relief of its obstructive effects marked the initial phase of this rapidly growing field. Whereas commissurotomy for mitral stenosis was performed with hesitancy and reticence in 1948 and 1949,[1, 2] this operation is now regarded by physician and surgeon alike as a routine procedure, the model for acquired valvular surgery. As its performance has become more universally applied, increasing numbers of physicians now have a personal experience with the problems which these patients present. A natural concern has developed as to the ultimate outcome of patients so treated. Many physicians have witnessed the dramatic and almost unbelievable results of this surgery in patients for whom there seemed no possible medical hope. On the other hand, these same physicians have encountered a number of patients who have not responded to the same degree and, by focusing their attention on these, have found their initial enthusiasm favorable but somewhat dampened.

A second but infinitely smaller group of internists have steadfastly refused to believe the reports of patients who describe their improvement in glowing terms primarily because, in many instances, they cannot substantiate this functional improvement by objective means. Therefore, in an attempt to clarify this state of confusion, it seemed imperative to review exhaustively a representative number of patients consecutively treated by commissurotomy five or more years ago, this span of time representing the longest available period of observation. The purpose of this communication then is to present in a detailed and painfully statistical manner all available data concerning fifty such patients, the first of whom was operated upon in January of 1949. It must be stated in advance, however, that more than average difficulty was encountered in obtaining all the information considered essential for this analysis because these patients live in many parts of the country. Nevertheless, over half the patients have been examined personally (O. H. J. and R. P. G.) within the past two months and complete information including electrocardiographic tracings and teleoroentgenograms has been obtained from

From the Departments of Thoracic and Cardiac Surgery of Presbyterian and Episcopal Hospitals, and the Department of Thoracic Surgery, Hahnemann Medical College and Hospital; Department of Cardiology, Presbyterian Hospital, and Department of Medicine, Hahnemann Medical College and Hospital.

Read at the Thirty-fifth Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, N. J., April 24 to 26, 1955.

both patients and attending physicians in the remainder during the same period. To ensure a completely uniform method of reporting operative findings all patients in this series have been operated upon by a single surgeon (R. P. G.).

ANALYSIS OF PATIENTS

Fifty-one patients were subjected to surgery five or more years ago. The first of these is now six and one-half years postoperative. One patient in the series was followed for the first two postoperative years only and at that time her condition was excellent, asymptomatic without medication. She has been deleted from this study, for her present status and whereabouts are unknown. Of the remaining fifty, thirty-six were women and fourteen were men. The average age at the time of surgery was 34 years.

Status Prior to Admission.—There was a definite history of rheumatic infection in twenty (40 per cent) of these patients. Each of the fifty patients was suffering from some degree of functional incapacity. There was a history of gross hemoptysis in twenty-three patients (46 per cent). Twenty-seven patients (54 per cent) had suffered one or more bouts of congestive failure. In six (12 per cent), arterial emboli had occurred from which a reasonable recovery had been made.

Admission Findings.—When admitted for preoperative and operative treatment to our hospital services, thirteen (26 per cent)of these patients were in gross chronic congestive heart failure. Nineteen (38 per cent) were in permanent atrial fibrillation.

Preoperative Classification of Patients.—As the mere presence of a mitral diastolic murmur in an asymptomatic patient was not then and is not now an indication for mitral commissurotomy, none of these patients was in Stage I (murmur without symptoms). Five patients (10 per cent) were suffering from fatigue and exertional dyspnea but as these symptoms had not progressed materially in the preceding year they were classified as Stage II (statically incapacitated). In thirty-six patients (72 per cent), in spite of continuous and vigorous medical management, a progressively downhill course was apparent (Stage III, progressively incapacitating). The remaining nine patients (18 per cent) showed constant evidence of congestive failure and as such were classified as Stage IV. Each of these patients was subjected to the standard mitral commissurotomy as described in previous publications[2-4] (Table I).

TABLE I. FUNCTIONAL CLASSIFICATION
(41 Living Patients)

PREOPERATIVE	POSTOPERATIVE			
Stages	I	II	III	IV
One (0)	0	0	0	0
Two (5)	2	3	0	0
Three (29)	11	11	6	1
Four (7)	1	4	1	1
Total (41)	14	18	7	2

Key: Written numbers designate preoperative functional status; Roman numerals designate postoperative functional status; Numbers in parentheses designate total number of patients per stage classified preoperatively.

Mortality.—Three patients died within the first postoperative month, representing an operative mortality of 6 per cent. Each of these was an advanced Stage III problem and each was in atrial fibrillation.

The first died suddenly from acute cardiac decompensation (the day before his proposed hospital discharge). Post-mortem examination revealed a moderately calcified valve of very limited pliability. The previous mitral insufficiency had been considerably aggravated by the posteromedial commissurotomy which had been performed because there was no anterolateral commissure, the mitral orifice being eccentrically placed. A moderately severe but previously undiagnosed aortic stenosis was noted. The second patient died four weeks after surgery, following a stormy postoperative course complicated by slowly increasing congestive failure to the point of anasarca. The valve had been opened by finger pressure alone which had caused an inadvertent tear of the posterior mitral cusp along its lateral attachment. Death was therefore caused by the production of too much mitral insufficiency clearly verified by post-mortem examination. This examination likewise revealed that the superior one-third of the left atrial lumen was completely obliterated by a thrombus the size of a lemon. The third patient died two hours after surgery, from which he never responded. His left atrium had been entered with difficulty through a completely obliterated appendage and the tiny valve orifice split by finger pressure alone. Autopsy revealed that the commissurotomy had been performed properly in the anterolateral commissure without damage to either leaflet. The cause of death was apparently due to operative and anesthetic shock. Aortic valve disease of the insufficiency type was found to be present.

Six additional patients died, one at six weeks, one at seven months, two at two and one-half years, and two at three years following the performance of commissurotomy. Three of these patients had been classified as Stage IV and three were in advanced Stage III. All these patients had extensively calcified valves whose leaflets were thickened, fibrosed, and nonpliable. The three Stage IV patients were in a terminal state at the time of surgery and their deaths are contributable to irreversible cardiopulmonary, hepatic, and renal disease. In a fourth, a preexisting mitral insufficiency was aggravated by surgery and this patient went downhill slowly as is their wont. The remaining two patients had completely inoperable valves due to marked calcification and unyielding induration precluding other than an inadequate commissurotomy. By and large, it can be stated that the cardiovalvular status of each of these patients had advanced to such a degree of deformity that no surgical intervention could be expected to be of lasting value.

Thus, there was an operative mortality of 6 per cent (three patients) and a late mortality within the first three postoperative years of 12 per cent (six patients). The remaining forty-one patients are living today, approximately five to six and one-half years postcommissurotomy and the data on these patients will comprise the remainder of this presentation.

Present Classification of 41 Living Patients.—(a) Each of the five patients originally classified as Stage II has lived a relatively normal life to date. Two of these are completely asymptomatic and can now be classified in Stage

I, for some slight element of their original diastolic murmur remains. The three others have been (conservatively) left in Stage II, for at times they have experienced more fatigue than that which could be considered normal and on a few other occasions their exercise tolerance was less than that which we expect in a normal person. (b) Twenty-nine of the thirty-five patients originally classified as Stage III are living. Eleven of these are completely asymptomatic and have reverted to Stage I. Eleven additional patients have been placed in Stage II, and six remain in Stage III although they continue to be functionally improved over their preoperative state. The remaining patient in this group has now been classified in Stage IV, for some element of chronic congestive failure remains constant. (c) Quite surprisingly seven of the ten patients originally classified Stage IV are living. One is completely asymptomatic and is in Stage I, four have been reclassified as Stage II, one is in Stage III, and one remains in the original classification, Stage IV.

To recapitulate, then, in the entire group of forty-one living patients there are now fourteen in Stage I, eighteen in Stage II, seven in Stage III, and two in Stage IV. It is readily apparent, therefore, that only one patient in the series is now classified at a higher level of incapacity than that prior to surgery. This rearrangement of the classification of these patients is purely didactic, was made by us, and is an attempt to evaluate patients purely within the limits provided by the original arbitrary stages as published in the past[5, 6] (Table I).

The Patient's Classification of His Own Present Status.—(a) On close and detailed interrogation of the patients themselves, confirmed by their home physicians, twenty of these forty-one patients (40 per cent of the original fifty cases) consider themselves to be in an excellent state of health. They deny symptoms during the course of all normal activity, although a few will admit to some minor limitations should they exert themselves considerably beyond their normal routine of life. These patients engage in such forms of exercise as golf, dancing, swimming, bicycle riding, and bowling. Eleven of them use no medication of any type, and are on a normal diet. Nine have continued to use digitalis sporadically. When questioned carefully on this point, their use of digitalis is in small dosage and on an average of every second or third day. By classification three of these patients were originally in Stage II, thirteen in Stage III, and four in Stage IV.

(b) Sixteen (32 per cent of the original fifty cases) consider themselves very definitely improved over their preoperative state. These patients feel that they are living a relatively normal life so long as they stay within certain self-imposed limitations which have been found by trial and error to be proper for them personally. Thirteen of these take a daily maintenance dose of digitalis but three require no cardiac medication. A few of these remain on a low salt diet to which they have long become accustomed, as they have stated, "I no longer have any desire for salt." Four use Diamox occasionally.

(c) Five of these forty-one living patients (10 per cent of the original fifty) regard themselves as unimproved over their preoperative state, although each passed through a period of two to four years during which he was con-

siderably improved. They have all rather profusely expressed gratitude for the surgery as performed and have stated, possibly euphorically, that without surgery they would not now be alive. By classification, four of these patients were Stage III and one in Stage IV prior to surgery.

To summarize and to compare these findings, the status of these forty-one living patients can be stated as follows: According to their present classification as rated by us, thirty patients (60 per cent of the entire group subjected to surgery) are better after five years than they were preceding surgery. According to the patients themselves, 72 per cent (thirty-six of the original fifty) are definitely better after five years than they were prior to surgery.

Analysis of Murmurs.—(a) Twenty-five of the forty-one living patients were considered on clinical auscultatory grounds to be cases of pure mitral stenosis. Seven of these now have no mitral diastolic murmur and three of these seven have no murmurs at all in any of the valve areas. Three others have a soft systolic mitral blowing murmur indicative of some insufficiency, and one has an additional soft diastolic murmur along the left sternal border suggestive of aortic insufficiency.

Eighteen of the twenty-five still have some degree of their original mitral diastolic murmur and ten of these have an additional systolic element that was not formerly present.

(b) Ten of the forty-one living patients originally had, in addition to their greatly predominant mitral diastolic murmurs, an element of mitral insufficiency as indicated by a systolic component to the murmur.[7] Six of these still have a measure of both diastolic and systolic murmurs. In three others the diastolic murmur has disappeared and is now replaced by a systolic murmur only. The one remaining patient has no detectable murmurs at this time.

(c) Four of the forty-one living patients originally had, in addition to their predominant mitral diastolic murmurs, a diastolic murmur along the left sternal border suggestive of aortic insufficiency. This was presumed to be of adynamic proportions, for no other clinical signs were present to substantiate a significant degree of aortic insufficiency. Each of these four cases still retains some measure of his mitral stenotic and aortic insufficiency murmurs. Two of these have obtained excellent functional results, one is markedly improved, and the other remains unimproved.

(d) One of the forty-one living patients originally had both a mitral systolic and diastolic murmur and an aortic diastolic murmur, and elements of all of these murmurs are still present although the patient is very definitely improved functionally. One additional patient of the forty-one living cases had systolic and diastolic murmurs both in the mitral and aortic areas and still has, but is likewise in an improved functional state.

To summarize the auscultatory findings in these patients, only four of these forty-one living patients have a completely murmurless heart at this time. Eleven have lost all evidence of a mitral diastolic murmur. Fourteen patients now have a systolic murmur at the apex in the mitral area of various degrees which they did not have prior to surgery.

From a functional standpoint those patients who had a pure murmur of mitral stenosis prior to surgery have had the best results. Fourteen of these twenty-five patients are now excellent, nine are improved, and two are unimproved. This result is a little better than that obtained in the ten patients who were predominantly mitral stenosis cases but who had an associated systolic component to the murmur prior to surgery. In this group four obtained an excellent result, four were improved, and two were unimproved.

Electrocardiographic Changes.—

Rhythm: There were twenty-nine patients in normal sinus rhythm at the time of surgery. Not infrequently atrial fibrillation appeared in this group during the early postoperative period, usually within the first four days. In nine of these incidences this arrythmia has remained to the present time, even though intensive quinidine therapy was carried out both during the hospital stay and on one or more occasions thereafter. All these patients, including those who are now in atrial fibrillation, have remained improved or excellent to the present time.

In the remaining twelve cases atrial fibrillation was present preoperatively and has remained constant to date. In this group five are unimproved over their preoperative state but the other seven maintain an improved to excellent status.

The P wave: In general, the P wave did not change either in its duration or configuration. Occasionally, the high voltage of the P wave, usually noted in cases with sustained and marked pulmonary artery hypertension, was reduced in amplitude after an adequate commissurotomy.

The QRS complex: There were ten cases in which, from the QRS standpoint, preoperative electrocardiogram was normal. This state remained essentially unchanged after commissurotomy except for slight counterclockwise rotation around the longitudinal axis.

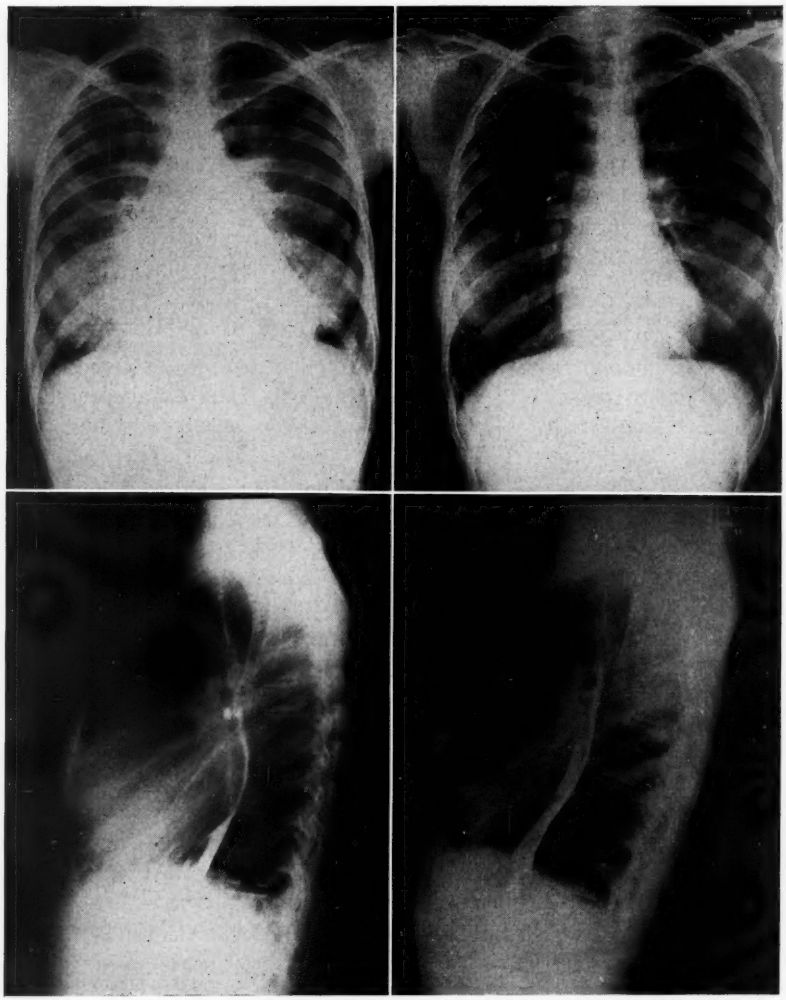
The remaining thirty-one showed moderate to marked right ventricular hypertrophy. This pattern showed a variety of changes. In eleven a recession of the right ventricular hypertrophy pattern was observed and in seven of these it completely disappeared.

In ten cases Lead V_1 showed a delay in the activation time of 0.4 to 0.6 second. These alterations returned to normal within one to two years after commissurotomy and have remained so to the present time.

In the remaining ten cases there was no significant change in the right ventricular hypertrophy pattern except for slight evidence of a combined hypertrophy pattern in one. In this individual a dynamic mitral insufficiency is now present.

Roentgenographic Findings.—Since the inception of this surgical program, the anticipated roentgenographic changes have always been the source of considerable speculation because of the graphic nature of this type of study.[8] It is frequently rather difficult to evaluate the observed changes in cardiac size due to the many postoperative variables such as pleural reaction, pericardial

reaction and adhesions, changes in the patient's weight, and the variations in x-ray techniques which occur even in the same institution at different times. Despite these difficulties the following statements can be made with confidence.



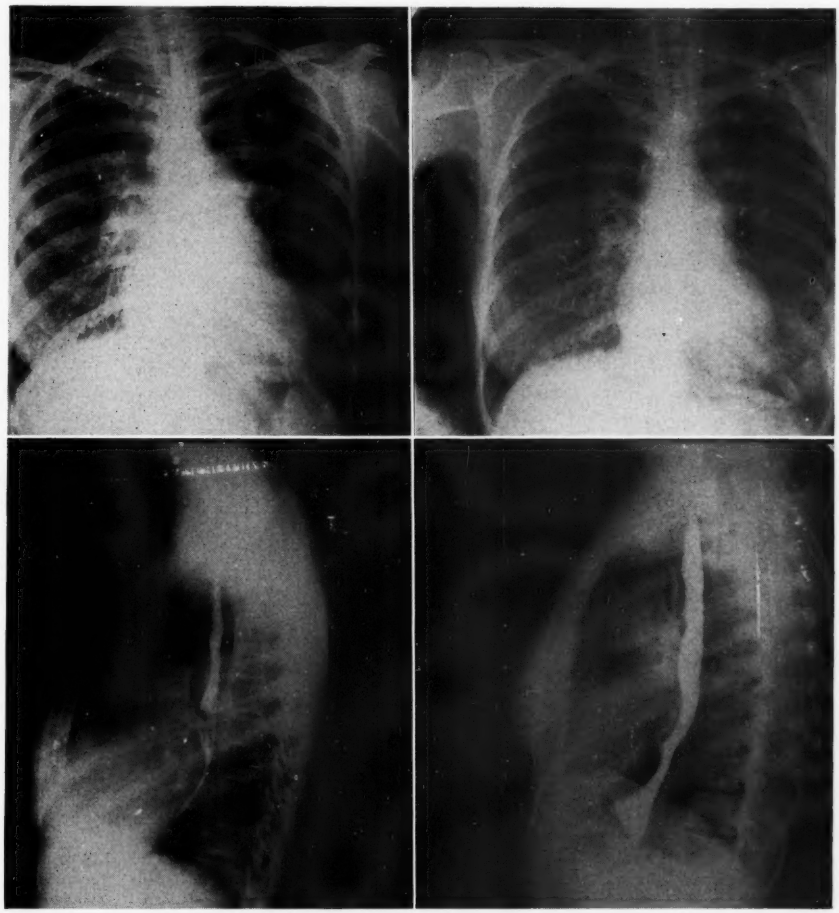
A. *B.*

Fig. 1.—E. I. Commissurotomy, Nov. 9, 1950. X-ray films taken (*A*) before surgery (Oct. 12, 1950) and (*B*) approximately five years thereafter (Feb. 16, 1955). This is the most dramatic diminution of heart size in this series. Twenty-four per cent of the living patients now have a smaller heart than before surgery.

In ten patients (24 per cent) the over-all heart size was obviously smaller (Fig. 1). This, of course, resulted from the diminution in size of the right ventricle, pulmonary artery, left atrium, and appendage. Nine of these

patients, as one would expect, have obtained an excellent result and the tenth is considerably improved (Figs. 1 and 2).

In five (13 per cent) the over-all size of the heart was obviously larger. This enlargement for the most part was reflected in an increased size of the left



A. *B.*

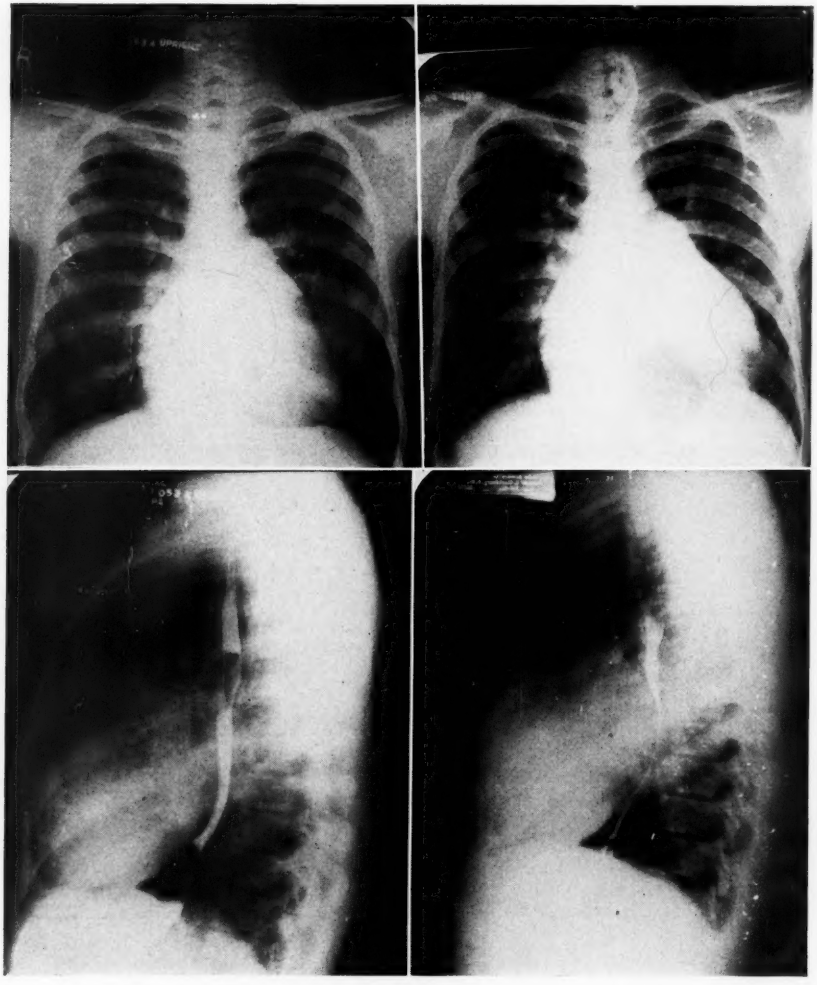
Fig. 2.—V. S. Commissurotomy, June 27, 1949. The usual degree of diminution in cardiac size noted in ten patients (24 per cent) whose hearts are now smaller than they were before surgery. *A*, June 21, 1949; *B*, Feb. 15, 1954.

ventricle and possibly the left atrium. Rather paradoxically perhaps, four of these patients consider themselves functionally improved and one is unimproved (Fig. 3).

In the largest group, twenty-six patients (63 per cent), there may be room for rather heated discussion. In our opinion these hearts, or at least these cardiac roentgenographic silhouettes, remain unchanged. There are many

who would argue both ways (larger or smaller) in this group, but careful study has indicated that the over-all cardiac mass is essentially unchanged (Fig. 4).

The implication of these findings must be very obvious. Whereas in the years immediately preceding surgery all these patients were showing progressive

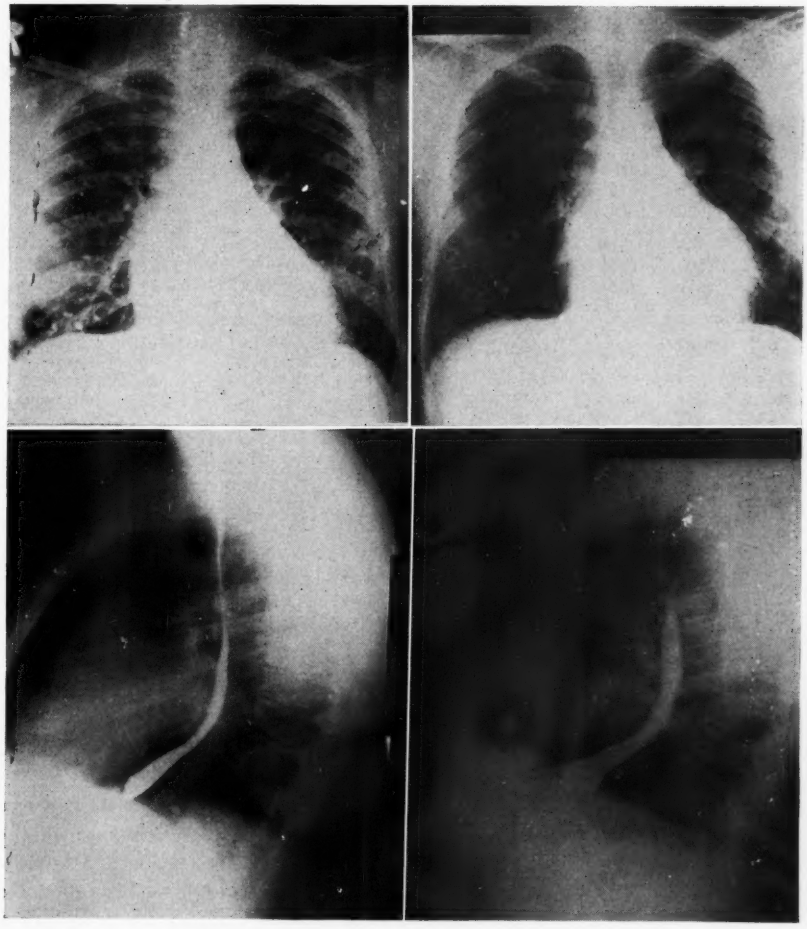


A. *B.*

Fig. 3.—J. K. Commissurotomy, Nov. 23, 1949. In five patients (13 per cent) there was some increase in the cardiac silhouette usually to the degree illustrated above. *A*, Nov. 14, 1949; *B*, March 9, 1954.

enlargement of their cardiac chambers, now and very significantly, 87 per cent of these hearts are either the same size or smaller. This is rather powerful evidence of an objective nature to show that surgery has affected the clinical course of these patients favorably.

Cardiac Catheterization Data.—It is extremely unfortunate that the catheterization studies in this important series of patients could not have been more detailed and complete. The preoperative studies were made at a time (five to six and one-half years ago) when our facilities for this type of examination were limited and primitive by present-day standards. Nevertheless, all data



A. *B.*

Fig. 4.—T. S. Commissurotomy, Nov. 7, 1949. In twenty-six patients (63 per cent) the cardiac roentgenographic silhouette indicative of over-all cardiac mass remained essentially unchanged. *A*, Nov. 1, 1949; *B*, March 3, 1954.

obtained are presented in Table II. Twenty-three of the twenty-seven cases in which data are available have obtained functional improvement of significant degree sustained to the present time. In all but two of these, the reduced pressure within the pulmonary vascular bed and right ventricle has paralleled

and objectively corroborated the clinical improvement. In these two (No. 11, H. E., and No. 12, R. B.), both of whom are in excellent condition today, the pressure drop was slight in one and the pressure was higher in the second. As the postoperative catheterization in these was carried out only one month after surgery, it would seem that enough time had not elapsed for the secondary circulation to readjust to its final levels.

TABLE II. AVAILABLE CATHETERIZATION DATA
(27 of 41 Living Patients)

CASE	PREOPERATIVE		POSTOPERATIVE		CLINICAL	
	RT. VENT.	PUL. ART.	RT. VENT.	PUL. ART.	MONTHS	RESULTS
1. J. B.	90/17 (41)	—	48/3 (18)	—	20	Excellent
2. E. W.	82/2	84/38	56/1	63/19	1	Unimproved
3. V. S.	141/5 (50)	143/56 (85)	33/14 (20)	36/15 (22)	15	Excellent
4. T. S.	43/3 (16)	42/12 (22)	27/5 (12)	—	33	Excellent
5. J. K.	60/4	81/31	23/8	24/6	65	Improved
6. S. G.	42/?	55/7	7/4	—	1	Improved
7. E. M.	55/36 (42)	49/30 (36)	28/0 (9)	35/10 (18)	13	Improved
8. V. C.	100/9 (39)	130/59 (83)	26/5 (12)	24/8 (13)	21	Excellent
9. G. K.	50/2 (18)	48/24 (32)	47/4 (18)	52/23 (33)	10	Unimproved
10. W. F.	25/–1	24/11	—	—	—	Unimproved
11. H. E.	66/3	54/27	48/8	48/21	1	Excellent
12. R. B.	36/1	31/15	54/4	—	1	Excellent
13. G. D.	89/0	87/39	50/–2	50/18	9	Improved
14. F. L.	68/0 (23)	—	52/2 (21)	—	16	Excellent
15. H. D.	72/5	78/42	—	—	—	Improved
16. C. W.	37/4	32/14	—	—	—	Unimproved
17. A. N.	(37)	(72)	(2)	(14)	61	Improved
18. M. S.	43/3	48/18	—	—	—	Improved
19. E. C.	64/–3 (25)	75/27 (43)	30/0 (10)	30/10 (17)	14	Excellent
20. J. A.	—	57/44	24/30	38/18	12	Excellent
21. E. I.	93/6 (35)	105/24 (51)	24/–6 (7)	23/4 (14)	61	Excellent
22. A. G.	59/–7	59/17	—	—	—	Improved
23. M. Q.	21/7	36/–3	—	—	—	Excellent
24. M. D.	32/–2	30/16	20/8	20/6	14	Excellent
25. H. O.	67/0	68/29	—	—	—	Improved
26. M. J.	78/5	82/35	—	—	—	Excellent
27. I. J.	53/0	63/26	48/0	36/15	1	Improved

In the four patients who remained unimproved after surgery, only two were catheterized during the postoperative period but neither of these showed a significant pressure drop, again showing rather close correlation to the observed clinical state.

Valvular Findings.—It may be stated categorically that in a tight mitral stenosis (an orifice reduced to a size of a cigarette or less), the more pliable the valve the better the result. A preoperative demonstration of valvular calcification has often conditioned the surgeon to be cautious in his prognostic statements to the prospective patient. Some degree of calcium from a fleck to complete replacement of all valvular tissues was present in twenty (49 per cent) of these patients. Although seventeen of these have remained in a definitely improved state to the present time, only six can be considered in excellent functional condition.

In those twenty-one patients (51 per cent) in whom there was no evidence of valvular calcification, eighteen remain vastly improved to date but, in contrast to the group with calcification, fourteen of the eighteen are considered

to be excellent, a very decided improvement. Even so, there were three with densely fibrotic valves without detectable calcium who are essentially unimproved over their preoperative state.

Reactivation of the Rheumatic State.—One always hesitates to make emphatic and far-reaching statements concerning the presence or absence of rheumatic activity in a patient who does not show the full-blown syndrome of rheumatic fever in all its aspects. This is a condemnation of the medical profession as a whole rather than a reflection on any of its members for such a diagnosis must be made on clinical impression and experience alone, as no definitive or diagnostic tests for specific recognition exist. One of us (O. H. J.) was the first to call attention to the possible reactivation of the rheumatic state following surgery and termed it "pleuropericarditis" for this is the common, early presenting symptom-complex of this syndrome.[9-11] Despite an attempt to maintain a conservative attitude toward the existence of this complication, it must be stated that in eight of these forty-one living patients there have been repeated postoperative episodes certainly to be regarded, even without laboratory verification, as rheumatic activation. This represents an incidence of 19.5 per cent. The clinical manifestations of these rheumatic bouts have included at one time or another all the time-honored stigmata of this disease such as cyclic fevers, painful swollen joints, tachycardia, pulmonary and peripheral congestion, and thoracic or precordial pain. When one appreciates that this surgery is being carried out in a smoldering bed of rheumatic disease, the more significant fact is that all patients do not show evidence of increased rheumatic activity. Be that as it may, the fact remains that rheumatic activity does occur after surgery just as it does from time to time throughout the life of any rheumatic patient, whether surgery has been performed or not. It is of interest that in only three of the eight patients with postoperative rheumatic activity was there evidence of rheumatic change in the biopsied left auricular appendage. Thirteen other appendages of these forty-one living patients likewise showed rheumatic stigmata but with no clinical evidence of rheumatic activity.

Thromboembolic Data.—Despite the fact that the left auricular appendage was traversed in each instance, seven were obliterated by thrombotic material, and that approximately half the valves contained calcium, only one operative embolus was produced. This resulted in a complete left hemiplegia from which the patient, by dint of constant perseverance and adherence to a well-managed program of rehabilitation, has obtained complete recovery except for the finer motions of the left hand. Clinically this patient has obtained an excellent result and is living a normal and useful life. This low incidence is in some contrast to the embolic incidence as found in our first 500 consecutive commissurotomies.[12] The over-all embolic incidence in this larger series was 4.4 per cent, of which 2.6 per cent were cerebral and the remainder peripheral. There have been no embolic episodes during the postcommissurotomy period to date.

Miscellaneous Postcommissurotomy Medical Events.—To further attest the improvement obtained and maintained by these patients, it is of interest to note

that a considerable number of these patients have undergone unrelated surgery and other medical events, in some instance of a rather serious nature. In each instance these have been tolerated without complication or deterioration of their cardiac status. Four patients have undergone normal pregnancies and deliveries;[13] in one instance two such episodes, without mishap to either mother or child. Two cholecystectomies have been performed; two appendectomies, one of which was for a ruptured appendix; one hysterectomy and one uterine myomectomy; three curettages of the uterus; one hemorrhoidectomy; and one patient survived a rather severe myocardial infarct from which he is now recovering nicely. As stated, all these operations and their attendant anesthetics were tolerated without evidence of cardiac disability.

SUMMARY AND CONCLUSIONS

1. The first fifty consecutive patients undergoing mitral commissurotomy approximately five to six and one-half years ago have been subjected to a detailed analysis in an attempt to ascertain their present subjective and objective status.

2. None of these patients was in Stage I (asymptomatic), five (10 per cent) were in Stage II (statically incapacitating), 36 (72 per cent) were in Stage III (progressively incapacitating), and 9 (18 per cent) were in Stage IV (terminally incapacitating).

3. There was an operative mortality of 6 per cent (3 patients) and subsequent death, 6 weeks to 3 years later, occurred in 12 per cent (6 patients).

4. The remaining 41 patients (82 per cent) are living and these have formed the basis for the following conclusions.

5. After a comprehensive study of each living patient, including an appraisal of the patient's subjectively reported clinical state as well as a correlation of the clinical findings, electrocardiographic tracings, teleoroentgenograms, and catheterization data, we conservatively conclude that 30 patients (73 per cent of those living or 60 per cent of the original 50) are in better condition and living a more nearly normal life than they were prior to surgery.

6. According to the patients' and their family physician's own appraisal of the present clinical status of these 41 living patients, 36 (88 per cent of those living, or 72 per cent of the original 50) are better after five years than they were prior to surgery (Fig. 5).

7. If one were to judge the present clinical status of these patients on the basis of their murmurs alone, it would be impossible to come to any definitive conclusions. Only four of these 41 living patients have a heart free of murmurs. Eleven have lost all evidence of their original mitral diastolic murmur. Fourteen patients now have a mitral systolic murmur of varying degrees which was not present prior to surgery. Those patients who have a pure murmur of mitral stenosis indicative of a small mitral orifice (cigarette size or less) routinely obtain the best results from mitral commissurotomy.

8. Although the electrocardiographic changes following surgery do not routinely show conclusive evidence of improvement, in a considerable percentage of patients changes are present indicative of a definitive return toward the normal.

9. Fluoroscopically and by teleoroentgenographic studies, ten (24 per cent) of the 41 living patients now have a cardiac silhouette which is obviously smaller than it was prior to surgery, 26 (63 per cent) have a cardiac mass which is of the same size as that noted before surgery, and in 5 (13 per cent) the heart size is obviously larger. The significant point rests in the fact that now, five or more years after surgery, 87 per cent of the patients have a heart the same size or smaller than it was before surgery, whereas in the years preceding surgery it was progressively becoming larger.

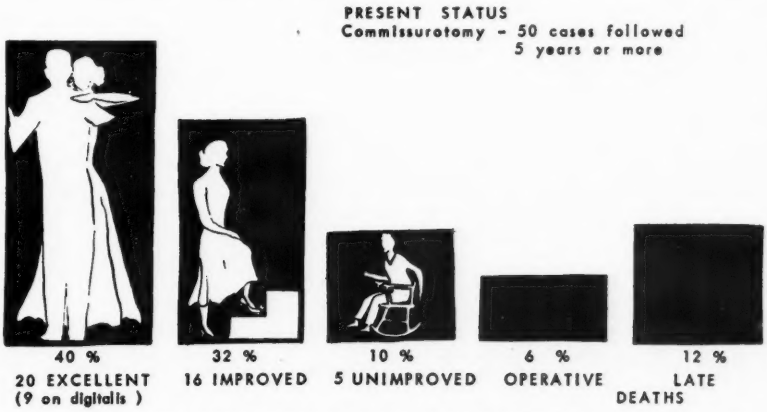


Fig. 5.—The present status of fifty cases in this series followed five or more years.

10. All available cardiac catheterization data parallel and corroborate the observed functional state of the patient whether he be improved or unimproved.

11. In 20 (49 per cent) of these patients valvular calcification was present and the degree of improvement in these, although quite satisfactory, was not as great as that seen in the 21 (51 per cent) cases whose valves were free of calcium deposits.

12. Definite evidence of rheumatic activity was observed during the postoperative period in eight patients (19.5 per cent).

13. No evidence of valvular restenosis has been observed in the 41 living patients nor in the valves of those who died in the postoperative period.

14. Only one operative embolus was produced in these 50 patients and that patient has obtained complete recovery of her left hemiplegia except for the restoration of the finer motions of the left hand. There have been no postoperative emboli to date (12 per cent prior to surgery).

15. There is no question in our minds but that commissurotomy has been of untold value to the majority of patients in this series and for the most part

that degree of improvement obtained in the first postoperative year has been maintained over a five-year period. There is no reason to suppose that it will not continue.

16. It is perhaps permissible to postulate that could the technical experience obtained in our over-all series of 740 commissurotomies have been applied to these first fifty consecutive patients the results might well have been considerably better.

REFERENCES

1. Glover, R. P., O'Neill, T. J. E., and Bailey, C. P.: Commissurotomy for Mitral Stenosis, Circulation **1**: 329-342, 1950.
2. Glover, R. P., O'Neill, T. J. E., and Bailey, C. P.: Mitral Stenosis—Its Surgical Treatment by Commissurotomy, Pennsylvania M. J. **53**: 375-384, 1950.
3. Glover, R. P.: The Technique of Mitral Commissurotomy. Presented at the Cardiovascular Symposium, Henry Ford Hospital, Detroit, March 17-19, 1955. Cardiovascular Surgery, Philadelphia, 1955, W. B. Saunders Company, pp. 179-202.
4. Glover, R. P., and O'Neill, T. J. E.: Surgery Of The Heart And Great Vessels, in Babcock's Principles and Practice of Surgery, ed. 2, Philadelphia, 1954, Lea & Febiger, pp. 950-1009.
5. O'Neill, T. J. E., Bailey, C. P., and Glover, R. P.: Observation on the Surgical Treatment of Mitral Stenosis by Commissurotomy, J. A. M. A. **147**: 1032-1033, 1951.
6. Janton, O. H., Glover, R. P., and O'Neill, T. J. E.: Indications for Commissurotomy in Mitral Stenosis, Am. J. Med. **12**: 621-625, 1952.
7. Janton, O. H., Heidorn, G., Soloff, L. A., O'Neill, T. J. E., and Glover, R. P.: The Clinical Determination of Mitral Insufficiency When Associated With Mitral Stenosis, Circulation **10**: 207-212, 1954.
8. Lehman, J. S., and Curry, J. L.: A Correlation of Roentgen and Surgical Findings in Two Hundred Cases of Rheumatic Mitral Valvular Disease, Am. J. Roentgenol. **71**: 599-610, 1954.
9. Janton, O. H., Glover, R. P., O'Neill, T. J. E., Gregory, J. E., and Froio, G. F.: Results of the Surgical Treatment for Mitral Stenosis (Analysis of One Hundred Consecutive Cases), Circulation **6**: 321-333, 1952.
10. Glover, R. P., O'Neill, T. J. E., Harris, J., and Janton, O. H.: The Indications for and the Results of Commissurotomy for Mitral Stenosis, J. THORACIC SURG. **25**: 55-77, 1953.
11. Soloff, L. A., Zaluchni, J., Janton, O. H., O'Neill, T. J. E., and Glover, R. P.: Reactivation of Rheumatic Fever Following Mitral Commissurotomy, Circulation **8**: 489-493, 1953.
12. Glover, R. P., Iaia, B. D., O'Neill, T. J. E., and Janton, O. H.: Thrombo-embolic Data in Mitral Stenosis Before and After Surgery, Proceedings of the Conference of the International Congress on Thrombosis and Embolism, Basel, Switzerland, 1954. Basel, 1955, Benno Schwabe & Co.
13. Glover, R. P., McDowell, D. E., O'Neill, T. J. E., and Janton, O. H.: Mitral Commissurotomy in Relation to Pregnancy, J. A. M. A. **158**: 895, 1955.

DISCUSSION

DR. DON L. FISHER, Pittsburgh, Pa.—Have any valvular lesions occurred postoperatively? Do you now follow the recent recommendation of the American Heart Association, to place all patients with rheumatic heart disease on penicillin permanently?

DR. ROBERT P. GLOVER (Closing)—To answer the question regarding the advisability of the postoperative prophylactic use of long-term antibiotics following mitral commissurotomy, our practice has been as follows: All patients have been advised to continue the use of oral penicillin, 200,000 units twice daily, for the first postoperative year. Thereafter, it has been suggested that this medication be taken during the winter months and at other times whenever symptoms of upper respiratory infection develop. We have had no way of checking the efficacy of this program but have felt that adherence to such a regimen might well check or minimize the recurrences of rheumatic episodes.

Quite probably most patients have not accepted this advice or have not carried out such a program over a long period of time. In any event, Dr. Janton, our cardiologist, has noted on a number of occasions, in our over-all series of 800 mitral commissurotomies, the appearance of murmurs in the aortic area not present prior to surgery. Presumably this may represent additional rheumatic valvular disease but whether this represents a manifestation of new pathology or merely a continued development of old pathology cannot be stated.

CONSIDERATIONS IN THE SURGICAL TREATMENT OF VENTRICULAR SEPTAL DEFECTS

EARLE B. KAY, M.D., HENRY A. ZIMMERMAN, M.D. (BY INVITATION), AND FREDERICK S. CROSS, M.D. (BY INVITATION)
CLEVELAND, OHIO

THE impression is gained from the literature, as well as from many of our medical and pediatric confreres, that ventricular septal defects are fairly benign, congenital cardiac malformations. It is not until one critically reviews a large number of his own cases, not only on clinical grounds but also by roentgen, cardiographic, and catheterization studies, that the fallacy of this impression becomes obvious.

Ventricular septal defects were first described by Roger[1] in 1879. He was impressed by what appeared to him to be the lack of objective signs and symptoms other than the finding of a harsh systolic murmur. He felt that it represented a lesion that was fairly well tolerated by the individual; yet, his conclusions were drawn on patients who were followed for relatively short periods of time in which there was no pathologic verification. These views on the benignancy of the condition, based on such incomplete evidence, have prevailed to the present time and are still quoted by leading texts on this subject. Abbott,[2] in 1932, reported 55 isolated ventricular septal defects, stating that symptoms were usually absent and that cardiac function was seldom disturbed. She also has been extensively quoted as to the benignancy of the lesion; yet, in 1936[3] she stated that the average age of death was 14.5 years, which does not bear out this impression. In her excellent text, Taussig[4] differentiates between the small (less than 1 cm.) type of defect described by Roger,[1] which presumably is well tolerated, and the larger, higher-placed type of defect in which the prognosis for the patient is more guarded. The complications of bacterial endocarditis and secondary changes in the pulmonary vasculature are mentioned, but the serious import of the latter complication is not really stressed.

With the more precise diagnostic method afforded by cardiac catheterization, these views are rapidly changing. This change in attitude should be further accelerated by the marked improvement shown in the few patients who have undergone surgery for closure of ventricular septal defects. Evidence demonstrating that ventricular septal defects may be a serious threat to life is now accumulating. All of the 24 patients whom we have studied have shown a considerable shunting of blood through the defect, and an elevated right ventricular and pulmonary artery pressure. In some cases it

From the medical and surgical services of St. Luke's and St. Vincent Charity Hospitals and also from the surgical laboratory of St. Luke's Hospital.

Supported in part by research grant from the Cleveland Area Heart Society.

Read at the Thirty-fifth Annual Meeting of The American Association for Thoracic Surgery at Atlantic City, New Jersey, April 24 to 26, 1955.

has been estimated that as much as 50 per cent of the left ventricular outflow is shunted into the right ventricle and pulmonary circulation. Others have confirmed such findings.[5] Of 34 patients who died with ventricular septal defects studied by Edwards,[6] 25 were infants who died under 1 year of age. Some of them had other causes of death. Lillehei[7] has reported an average pressure of 70 mm. Hg in the right ventricle in 29 patients operated upon. As noted in Table I, there appears to be a correlation between the percentage and duration of shunt flow and the right ventricular and pulmonary artery pressures; that is, the greater the shunt flow, the higher the pressure obtained in the right ventricle and pulmonary artery up to a certain point. When the pulmonary hypertension and pressure within the right ventricle begin equalizing the left ventricular pressure, shunt flow will diminish and might even reverse, indicating the severe changes in the pulmonary vasculature. Generally, there is a significant degree of arterial desaturation associated with the higher pulmonary pressures.

TABLE I. CARDIAC CATHETERIZATION FINDINGS ON TWENTY-TWO PATIENTS WITH VENTRICULAR SEPTAL DEFECTS

NAME	AGE	VOL. % O_2		PRESSURE (MM. HG.)		O_2 SAT. %
		RA	RV	RV	PA	
C. W.	3	13.79	15.50	44/8	40/14	93.4
D. S.	1.5	8.14	10.17	28/2		
J. B.	3.5	9.37	11.36	34/2	28/9	
K. H.	3.5	9.49	10.09	37/3		90.1
J. R.	4	10.52	11.12	34/0	34/7	94.0
L. F.	5	10.40	9.72	100/25	100/66	87.2
G. C.	5	9.90	14.20	80		89.2
J. B.	6	7.17	10.40	98/2	92/52	85.4
D. D.	6.5	9.35	12.82	94/12	94/40	94
G. G.	6.5	14.94	17.9	103/3	100/60	77.7
R. M.	7	12.3	14.23	38/2	38/8	97.5
S. V.	7	10.9	14.9	61/-5	52.4/20	87
D. L.	9	8.01	8.87	92/4	66/12	79.1
N. B.	11	11.1	11.24	116/12	116/76	83.1
B. W.	12	10.2	14.12	30/3		93.5
B. F.	15	10.4	13.33	36/2	26/12	94.8
U. D.	15	12.1	13.6	73/-12	68/25	90
M. F.	17	12.15	14.03	108/4	104/50	78.1
J. M.	19	17.3	20.3	55.5/-17	55/18.7	87
I. M.	20	14.27	15.9	80/3		95.8
V. L.	21	12.53	14.67	52/2	33/5	93.4
P.	52	11.9	13.45	78/0		65

Analysis reveals a correlation between the percentage and duration of shunt flow and the right ventricular and pulmonary artery pressures. Generally there is a significant degree of arterial desaturation associated with the higher pulmonary pressures.

Repeat catheterization studies performed on the same patients over a period of one to two years have demonstrated progressive elevation of the intracardiac and pulmonary artery pressures in most instances. In a few, the catheterization studies have not shown a significant increase over this period of time. In some patients with essentially static cardiac catheterization findings, a progression of clinical symptoms and general disability was noted. The pulmonary vascular bed is able to accommodate the increased flow through the lungs initially. Later, pulmonary hypertension, right ventricular hypertrophy, and medioscleros is of the pulmonary vessels develop (Fig. 1).

There has been some controversy as to the etiology of the pulmonary vascular changes. Edwards,[8-9] in particular, has postulated that these changes are an additional congenital defect similar to those noted in primary pulmonary hypertension. Dammann and associates,[10-11] on the other hand, feel that these changes are not congenital but are the result of long-standing pulmonary hypertension resulting from increased blood flow through the shunt. Unfortunately, these vascular changes from pulmonary hypertension may reach

Fig. 1.—Gross specimen illustrating the marked degree of pulmonary mediosclerosis associated with pulmonary hypertension secondary to a septal defect and increased pulmonary blood flow.

a stage where they are no longer reversible and surgery directed toward closure of the defect will either end in failure or do the patient little, if any, good.

There is no question about the desirability of closure of significant ventricular septal defects. The problem should be focused on the perfection of surgical techniques and the crystallization of the indications and contraindications for surgical intervention. One must be cognizant of the facts that, if benefit is to be derived by surgical closure, such patients should be operated

upon before irreversible damage to the pulmonary circulation develops. Furthermore, increasing pulmonary and right ventricular pressures add to the risk of surgical intervention. Of the 5 patients with ventricular septal defects operated upon by us, the average right ventricular pressure in the three patients who died postoperatively was 98.7 mm. Hg and their arterial oxygen saturation was 89.4 per cent, as compared to a pressure of 62 mm. Hg and an arterial saturation of 94.6 per cent in the two successfully treated patients. In our experience, infants and children with ventricular septal defects have shown evidence of elevated pulmonary pressures and failure at a much earlier age than those with atrial septal defects. Our studies have shown that when the ventricular pressure is over 90 mm. Hg, in the presence of arterial saturation of less than 90 per cent, pulmonary vascular changes that might not be reversible undoubtedly have taken place. Other factors, however, must be taken into consideration, such as the age of the patient, duration during which elevated pressures have existed, the size of the heart, the degree of pulmonary vascularization (by x-ray), and the pulmonary reserve. One must weigh the risks of operation against the dangers of waiting.

Patent ductus arteriosus, atrial septal defects, and ventricular septal defects, from a physiologic standpoint, represent essentially the same type of lesion, that is, one with left to right shunting of blood with overloading of the pulmonary circulation. The indications for surgery, with few exceptions, in patent ductus arteriosus have approached the ideal. When the diagnosis is made and the optimum age for surgery is reached, the operation is performed. Unless the patient is getting into severe difficulties, we follow essentially the same pattern with atrial septal defects. Many of our patients, however, first come to us well beyond this age group. Ventricular septal defects are handled somewhat differently. If the ventricular septal defect is tolerated fairly well on the basis of such clinical findings as exercise tolerance and accepted weight gain, and if the x-ray picture and cardiogram are not significantly abnormal or show no rapid progression, the child may be followed to an optimum time for surgical correction. If clinical symptoms and cardiographic and x-ray changes are severe, however, catheterization should be carried out early, and if intraventricular and pulmonary pressures are elevated, surgical repair of the defect must be given serious consideration.

TECHNIQUES

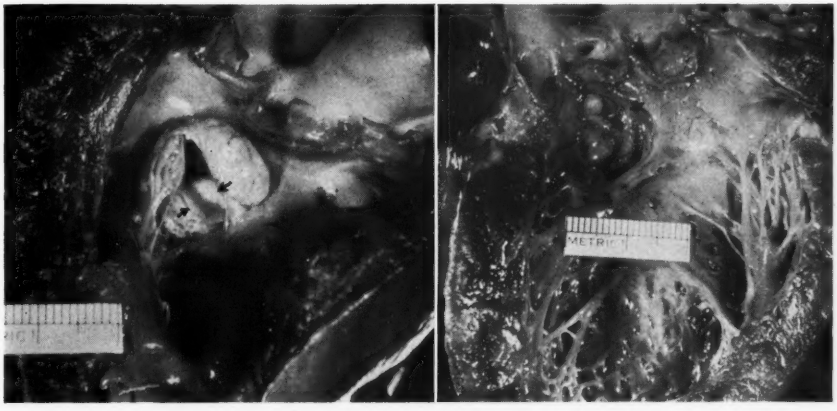
Early attempts at closure of ventricular septal defects utilized so-called "closed techniques." Murray,[12] in 1948, attempted to close ventricular septal defects by passing fascia lata strips in an anteroposterior direction through the ventricular septum and defect. In 1952 Bailey and his associates[13] employed transventricular pericardial grafts to plug the defect. However, such grafts tend to become atrophic, fibrotic, and ineffective. In 1953[14] we performed the first closure of a ventricular septal defect by suture technique in a 20-year-old white girl, employing a "closed technique." Although a murmur is still present, this patient has been significantly improved by operation

and now leads a normal life. Catheterization studies performed eighteen months postoperatively demonstrated a reduction in the right ventricular pressure from 80/3 mm. Hg preoperatively to 40/4 mm. Hg afterward. The oxygen studies preoperatively were 13 volume per cent in the superior vena cava, 12.5 volume per cent in the inferior vena cava, 14.27 volume per cent in the right atrium, and 15.9 volume per cent in the right ventricle. These changed postoperatively to 11.07 volume per cent oxygen in the superior vena cava, 12.21 volume per cent in the inferior vena cava, and 12.69 volume per cent in the right ventricle. The sample from the right auricle was in the region of the coronary sinus.

Swan[15] and Bailey[16] have attempted closure of ventricular septal defects by direct suture through the open right ventricle, employing hypothermia without success. Gibbon[17] has employed the open technique in a 38-year-old man, maintaining the cardiorespiratory functions by an extracorporeal circuit for twenty-six minutes. This patient died five hours postoperatively. The greatest success has been experienced by Lillehei, Varco, and their associates in employing cross circulation while closing ventricular septal defects through the open heart. Their latest report[7] revealed 22 successful closures and 7 postoperative deaths with this technique. During the past two years we have operated upon six patients with ventricular septal defects. One of these patients was an 11-month-old baby with tetralogy of Fallot. Four patients have been operated upon through an open cardiotomy. In three, cross circulation was employed and, in one, a pump with a reservoir of arterial blood was used. In only one patient was the operation successful. Two patients have been operated upon by "closed techniques"—one successfully, as reported previously, and the second unsuccessfully. This latter patient was a 5-year-old girl with pulmonary pressures of 100/60 mm. Hg and an arterial saturation of 89 per cent. The catheter apparently had withdrawn at the time of the ventricular sample and she was thought to have an atrial septal defect as well as a ventricular septal defect. The right auricle was explored first. The atrial septum was intact, but a ventricular septal defect could be palpated beneath the septal leaflet of the tricuspid valve. The defect was being closed by means of one of the "closed techniques" developed in the laboratory, which will be described later, when ventricular fibrillation developed. The heart could never be restarted after the ventricular fibrillation was finally stopped.

Regardless of the ultimate technique used, the surgeon must be cognizant of certain facts regarding the anatomy, pathology, and physiology of ventricular septal defects which dictate the precise way in which they must be closed. Ventricular septal defects are most commonly located in or just anterior to the membranous septum, just proximal to the aortic valve on the left ventricular side and adjacent to the septal leaflet of the tricuspid valve on the right ventricular side (Fig. 2). They are found less commonly in the muscular septum. This discussion will be limited to the high membranous defects. Occasionally, small defects may be bounded by a well-defined septal edge.

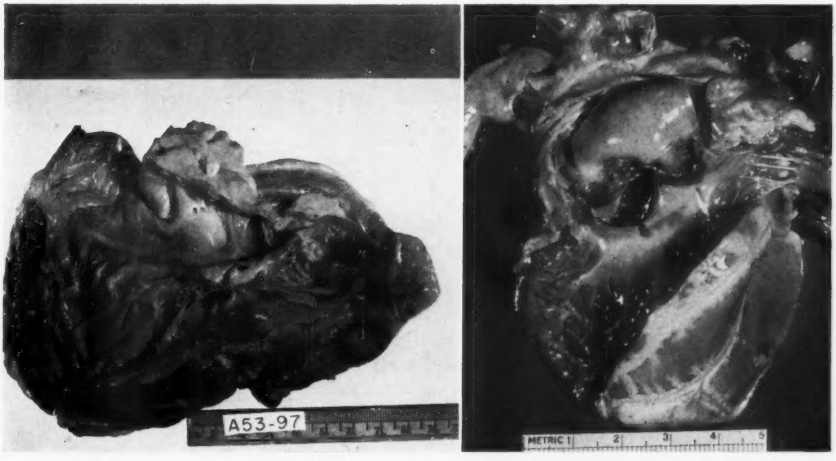
However, most of the patients who die from their septal defects or who have defects causing sufficient disability to warrant operation have larger defects, and there is little or no septal tissue on the upper border because of the failure



A. *B.*

Fig. 2.—*A*, Photograph of a large ventricular septal defect in a 5-year-old girl as viewed through the right ventricle. The arrow on the left is at the edge of the septal leaflet, while the one at the right overlies the anterior leaflet hypertrophied from the abnormal left to right shunt flow.

B, Defect from the same patient, as viewed through the left ventricle. Arrows demarcate the edges of the defect. The opening of the defect is filled with valvular leaflets from the tricuspid valve.

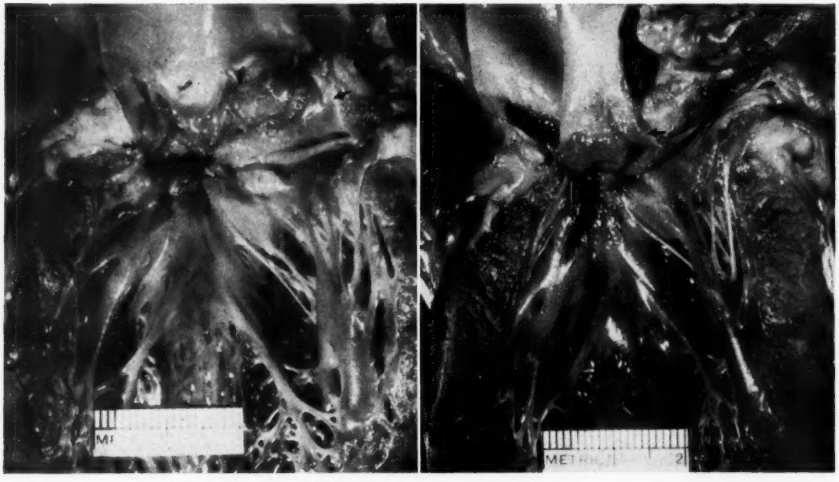


A. *B.*

Fig. 3.—*A*, Photograph of autopsy specimen from a 16-year-old girl who died of a ventricular septal defect. Defect is located beneath septal leaflet of tricuspid valve. Only the lower portion of the defect can be visualized extending beyond the septal leaflet. Arrows demarcate the upper border of the defect beneath the septal leaflet.

B, Photograph of autopsy specimen from a 1-year-old infant who died of a ventricular septal defect, which demonstrates the absence of septal tissue above at the aortic valve and the half circle contour of the defect.

of the aortic septum growing down to fuse with the ventricular septum. Instead of being round, such defects are in the shape of a half circle, the upper edge of which may be formed by the annulus of the aortic and tricuspid valves (Fig. 3). In some instances there is slight overriding of the aortic valve, so that, in essence, there is little tissue, if any, above to suture to without incorporating either the aortic or tricuspid leaflets. It is mandatory that such defects be closed in a longitudinal manner approximating the apical surface of the defect to the annulus, as demonstrated in Fig. 4. If such defects are



A. *B.*

Fig. 4.—*A*, Gross specimen of a ventricular septal defect viewed through the open left ventricle, demonstrating the correct apposition of the lower edges of the defect to the aortic annulus in a longitudinal direction. Defects closed in this manner do not occlude the aortic outflow tract or aortic valve. Edges of opened aortic valve are demarcated by arrows.
B, Same defect as above, demonstrating partial occlusion of aortic valve when ventricular septal defect is closed in a transverse direction.

closed in a transverse direction, it is impossible to close them completely without embarrassing the aortic outflow tract. It should be stressed that the defect, when viewed through the open right heart, is frequently covered by the crista supraventricularis and that this landmark must not be mistaken for the lower edge of the defect or used in its closure. Lillehei and his associates have accomplished closure by two one-half purse-string sutures. When there is some septal tissue above, this appears to be a satisfactory technique. In a number of specimens examined, however, either the septal tissue above is lacking or it is so concealed under the septal leaflet of the tricuspid valve that it is difficult or impossible to see and to close it without incorporating either an aortic cusp or part of the tricuspid leaflet, even though the valve leaflet is retracted and its papillary muscle severed. In many instances it appears that such defects, when lacking in septal tissue above, can be closed better by passing the suture from the lower edge out through the pliable myocardium

just proximal to the aortic valve at the junction of the aorta and right auricle which can be easily seen by retracting the right auricular appendage (Fig. 5). This is in a region free of the coronary vessels. This technique is particularly useful in operating upon patients with an overriding aorta, in which case the overriding and septal defect are closed at the same time. When such defects are closed in this manner there is no danger of incorporating either the aortic cusps or tricuspid leaflets. This technique can be employed in either open or closed myocardiotomies. The position of the incision into the right ventricle is also of importance. Unless it is necessary, in cases of associated pulmonary or infundibular stenosis, to make the incision along the pulmonary outflow tract, it should be placed in the direction of the right ventricular inflow tract so that significant coronary arteries are not severed with resulting myocardial ischemia.

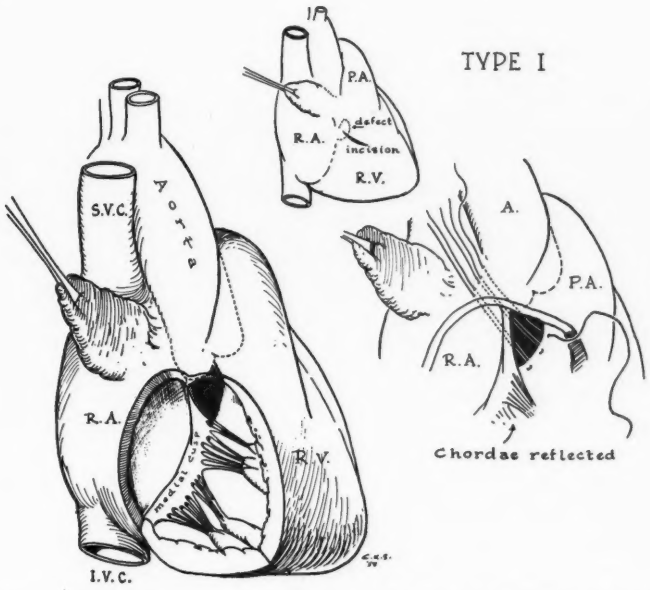


Fig. 5.—Diagrammatic sketch of Type I method for closure of ventricular septal defects. Finger is inserted into right ventricle, septal leaflet is retracted, and lower septal edge is palpated. A suture is introduced on a large curved, blunt needle and inserted through the myotomy opening, through the lower edge of the septal defect, and out through the angle between the aorta and right auricle. The other end of the suture is rethreaded and passed in a similar manner. Several sutures are placed and tied by the assistant as directed by the surgeon's intracardiac finger (see text).

In approximately ten months' experience with the use of controlled cross circulation, both in the laboratory and clinically, many problems have arisen, of which some are applicable to the cross-circulation technique per se and others are applicable to open cardiotomy using any type of artificial pump oxygenator.

Obviously, the critical aspect of the cross-circulation technique is the proper preoperative balance between the donor and recipient circuits and the

maintenance of this precise balance during the period of intracardiac surgery. To obtain this critical balance, we have continued to use the single-headed pump and have maintained equality of flow in both circuits through the use of special adaptors on each line.[18] By means of these, the tension, and hence the internal diameter of the rubber tubing within the pumping mechanism, can be varied; this, in turn, varies the output of the pump. Minute changes in flow can be obtained and balance of the two circuits within 1 c.c. in five minutes maintained. In spite of proper preoperative balance, imbalance during surgery may occur due to obstruction of either intake catheter, especially in the superior and inferior venae cavae. To minimize the incidence and the grave consequences of such a complication, blood reservoirs have been inserted in both circuits. The reservoirs are constructed to eliminate the marked negative pressure effect of the pump at the intake catheters which, previous to the use of the reservoirs, contributed to the collapse of the cavae about the intake catheter, thus throwing the circuits out of balance and leading to possible exsanguination of the donor. Instead of drawing blood directly from the recipient and donor, the pump now draws blood from the two reservoirs and a constant flow of blood to each person is assured by the maintenance of the blood level in the reservoir. In addition, with this system either person can be selectively transfused with a measured quantity of blood at a rapid rate. Through the incorporation of such a reservoir on the donor-to-recipient circuit, the variable effect of the blood pressure head in the donor on blood flow rates from donor to recipient has been eliminated.

Another source of imbalance of the two circuits may be the rapid loss of blood in the recipient, or operated person. Because of distortion of the aortic annulus in the open, collapsed heart, or because of the associated aortic valve defects, the regurgitation of blood through the aortic valve, and hence through the ventricular septal defect, may be so copious at times as to obscure the operative field completely. This regurgitation may be eliminated for a short period by the intermittent occlusion of the ascending aorta by an encircling ligature while stitches are being placed in the ventricular septal defect. It has not been established how well the diseased heart will tolerate such intermittent coronary inflow stasis. In the laboratory animal, it appears that the coronary arteries can be occluded safely up to two minutes. To compensate for these periods of coronary artery occlusion, we have been following such periods of ascending aortic occlusion by occluding the descending aorta in order to increase temporarily the pressure in the aortic arch and increase the coronary flow. It is felt that this additional boost of coronary blood following complete stasis has been beneficial. An alternate method of placing the ligature on the ascending aorta below the coronary arteries is being explored at the present time. This, of course, will allow continued coronary flow during the periods of aortic occlusion. Perhaps occlusion of the aorta in this area will hamper proper closure of the ventricular septal defect.

In spite of these added safeguards, we feel that it still would be beneficial to have an efficient, but not too complicated, flow meter incorporated in each

circuit so that the exact rates of blood flow in both persons would be known in any given instant. Such a flow meter is being developed at the present time.

A suitable donor was not available in 3 of the first 6 patients we considered for surgery utilizing cross circulation. Looking for a way to eliminate the donor in such instances, it was decided to investigate the possibility of substituting a large reservoir of oxygenated blood for the donor individual. In small patients with flow rates of 30 to 40 c.c. per kilogram per minute, 10 to 20 liters of oxygenated blood would be sufficient. This was investigated and found to be successful in the laboratory and was tried on one clinical case. This patient developed ventricular fibrillation as soon as the ascending aorta was occluded and a normal rate could not subsequently be restored.

At the present time the technique of controlled cross circulation is not applicable to adult patients requiring open intracardiac surgery. Possibly the use of oxygenated blood reservoirs can be utilized in spite of the fact that a large person will require a large volume of blood even at basal flow rates.

CLOSED TECHNIQUES

Because of the inherent difficulties previously discussed in the presently available open techniques, means of repairing ventricular septal defects by "closed techniques" are continuing to be sampled. All these techniques utilize the principle of approximating the pliable myocardial wall between the aorta and right atrium to the lower edge of the septal defect.

Closure of ventricular septal defects by the closed method are best approached through a right anterolateral incision through the third or fourth interspaces, with transection of the sternum if necessary. The superior and inferior venae cavae should be isolated and an encircling tape put in place to provide for temporary vena caval inflow stasis in the event of hemorrhage from the hypertrophied right ventricle or to allow temporary collapse of the right ventricle for easier approximation of the edges of the defect. Three variations of the closed technique have been employed experimentally and will be referred to as Types I, II, and III. A ventricular septal defect is first produced beneath the septal leaflet of the tricuspid valve, simulating as much as possible those defects found clinically. Such defects frequently interrupt fibers of the bundle of His and conduction arrhythmias usually result.

In Type I (Fig. 5), a purse-string suture is placed in the anterior border of the right ventricle in such a position that a large, blunt-nosed, curved needle can be introduced alongside the finger in the same opening. The intracardiac finger retracts the septal leaflet away from the defect. The end of the needle first engages the apical surface of the defect, care being exercised not to include either the crista supraventricularis or the chordae tendineae. The needle is then passed upward between the annuli of the tricuspid and aortic valves and exits through the myocardium proximal to the aortic valve at its junction with the right auricle. The right auricular appendage is retracted laterally. The other end of the suture is then threaded and passed in a similar manner, so that a mattress suture is so placed. A small piece of pericardium is placed on this suture and

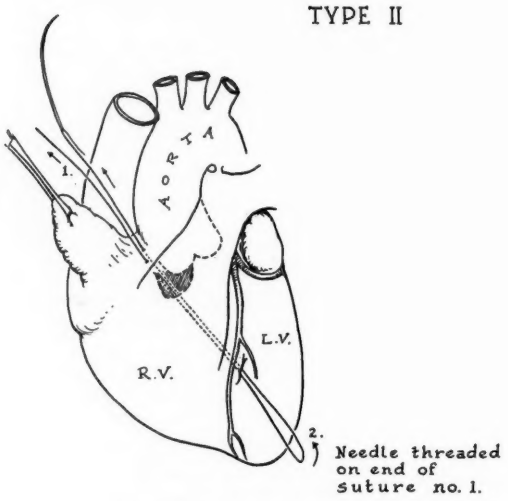


Fig. 6.—Diagrammatic sketch of Type II method for closure of ventricular septal defects. Similar to Type I except for introduction of suture along course of ventricular septum. Direction and position of suture is determined by the intracardiac finger (see text).

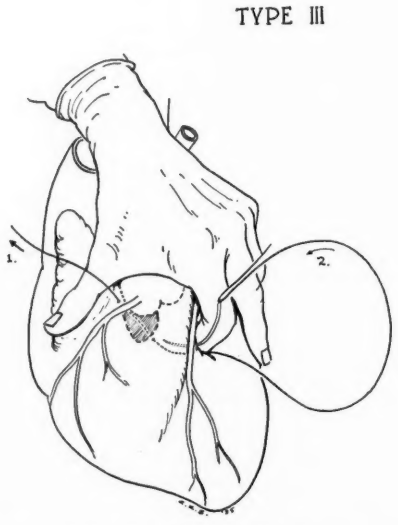


Fig. 7.—Diagrammatic sketch of Type III method for closure of ventricular septal defects. Similar to Type I except for the approach through the left ventricle rather than the right ventricle.

slid down to the septal edge, so that the stitch is less likely to tear out. Two to three such sutures appear adequate to close the defect. The sutures must be placed in a longitudinal manner, rather than transversely, so that the outflow tract of the left ventricle is not encroached upon. The sutures exit posterior to the right coronary artery. The assistant then ties the sutures outside the heart, over a piece of pericardium, the degree of tautness being determined and directed by the operator's intracardiac finger. This appears to be an effective way of closing such defects.

Type II (Fig. 6) consists of directing a suture through the ventricular septum. The needle is introduced just to the left of the descending coronary artery, and is directed by the intracardiac finger to engage the apical edge of the defect and to exit as in the preceding technique. The other end of the suture is threaded and passed in a similar manner. The anterior sutures are passed through the pericardium and the ends of the suture are tied over a section of pericardium to lessen the possibility of the sutures pulling through the myocardium. Because of the relationship of the direction of the suture in relation to the defect, there is a tendency for the suture to pass through the defect in a diagonal manner so that when the sutures are tied there may be some impingement on the aortic outflow tract. This is lessened the further toward the apex the sutures are introduced anteriorly.

In the Type III approach (Fig. 7), the ventricular septal defect is closed in a manner similar to Type I, but through a left myocardiotomy opening. Theoretically and experimentally, this appears to be a simpler approach, for one does not have to contend with the chordae tendineae and tricuspid leaflets on the left side. Practically, however, such an approach would be difficult because of the inaccessibility of the left ventricle in the presence of a hypertrophied right ventricle.

RESULTS

Although total experience in terms of patients has been small and the results in terms of survival have not been outstanding, the benefits to those involved in perfecting techniques for the closure of ventricular septal defects have been outstanding. To date, closure has been attempted in only 6 patients —2 by closed techniques and 4 by open techniques. The 20-year-old girl who underwent repair of a ventricular septal defect by closed technique in 1953 is living and well at the present time. Although a murmur is still present, her right ventricular pressure has dropped from 80/3 mm. Hg preoperatively to 40/4 mm. Hg postoperatively. The second patient in whom a closed technique was employed died of ventricular fibrillation at the time of surgery. Three patients have been operated upon utilizing the controlled cross circulation, with one successful closure. One patient died postoperatively after a successful closure and the other patient died at the time of operation from excessive regurgitation of blood through the aortic valve and septal defect, as described previously. There have been no donor deaths, although in the last case the donor suffered a large blood loss consequent to excessive blood loss in the

recipient. One patient was operated upon by the open method employing a pump and a reservoir of oxygenated blood. This patient had an extremely large defect in a markedly hypertrophied heart.

DISCUSSION

Obviously, the surgical repair of ventricular septal defects is not a closed chapter. The technique of controlled cross circulation and the substitution of a large reservoir of arteriolized blood in place of the donor individual probably offers the best approach to intracardiac surgery at the present time. The use of hypothermia has been disappointing. The perfection of an adequate pump-oxygenator probably will supply the ultimate answer. In the meantime, invaluable experience and knowledge are being gained by utilizing available techniques.

We feel strongly that the means of closing ventricular septal defects by closed techniques should receive continued investigation, for, if effective, they are generally simpler than open techniques. In such closed techniques, it is felt that the principle herein described, of reconstituting the membranous ventricular septum by inversion and approximation of the pliable cardiac wall between the right atrium and aorta to the apical portion of the defect, holds promise.

REFERENCES

1. Roger, H.: Communication congénital du coeur pa l'inocclusion du septum interventriculaire, Bull. Acad. méd. **8**: 1074-1094, 1879.
2. Abbott, M. E.: Congenital Heart Disease, in: Nelson: Loose-Leaf Medicine, New York, 1932, Thomas Nelson & Sons, vol. 4, p. 20.
3. Abbott, M. E.: Atlas of Congenital Cardiac Disease, New York, 1936, American Heart Association, p. 60.
4. Taussig, Helen B.: Congenital Malformations of the Heart, New York, 1947, Commonwealth Fund.
5. (a) Handelsman, J. C., Bing, R. J., Campbell, J. A., and Griswold, H. E.: Physiological Studies in Congenital Heart Disease. V. The Circulation in Patients With Isolated Septal Defects, Bull. Johns Hopkins Hosp. **82**: 615, 1948.
   (b) Dexter, L., Haynes, F. W., Burwell, C. S., Eppinger, E. C., Sosman, M. C., and Evans, J. M.: Studies of Congenital Heart Disease. III. Venous Catheterization as a Diagnostic Aid in Patent Ductus Arteriosus, Tetralogy of Fallot, Ventricular Septal Defects and Auricular Septal Defects, J. Clin. Invest. **26**: 561, 1947.
   (c) Baldwin, E. D., Moore, L. V., and Noble, R. P.: The Demonstration of Ventricular Septal Defect by Means of Right Heart Catheterization, Am. Heart J. **32**: 152, 1946.
6. Edwards, J. E.: Personal communication.
7. Lillehei, C. W.: Direct Vision Closure of Interventricular Septal Defect by Means of Controlled Cross Circulation, presented at International Symposium on Cardiovascular Surgery, Detroit, March, 1955.
8. Edwards, J. E.: Structural Changes of the Pulmonary Vascular Bed and Their Functional Significance in Congenital Heart Disease, 26th Hektoen Lectures, Frank Billings Foundation, 1950.
9. Edwards, Jesse E.: Functional Pathology of Congenital Cardiac Disease, Pediat. Clin. North America, p. 13, February, 1954.
10. Dammann, J. F., Jr., and Muller, W. H., Jr.: The Role of the Pulmonary Vascular Bed in Congenital Heart Disease, Pediatrics **12**: 307, 1953.
11. Dammann, J. F., Jr.: Symposium on Adjustments Between the Systemic and Pulmonary Circulation. Clinico-Anatomic Correlations, presented at International Symposium on Cardiovascular Surgery, Detroit, March, 1955.
12. Murray, Gordon: Closure of Defects in Cardiac Septa, Ann. Surg. **128**: 843, 1948.
13. Bailey, C. P., Lacy, M. H., Neptune, W. B., Weller, R., Arvanites, C. S., and Karasic, J.: Experimental and Clinical Attempts at Correction of Interventricular Septal Defects, Ann. Surg. **136**: 919, 1952.

14. Kay, E. B., and Zimmerman, H. A.: Surgical Repair of Interventricular Septal Defects, J. A. M. A. **154**: 986, 1954.
15. Swan, Henry: Personal communication.
16. Bailey, C. P.: Personal communication.
17. Gibbon, J. H., Jr.: Discussion of Paper, Controlled Cross Circulation for Open Intracardiac Surgery by Warden, H. E., Cohen, M., Read, R. C., and Lillehei, C. W.: J. THORACIC SURG. **28**: 331, 1954.
18. Jones, R. D., Kay, Earle B., Hudack, S. S., and Cross, F. S.: Suggested Modifications in the Application of the Sigmamotor Pump to the Technique of Controlled Cross-Circulation, J. THORACIC SURG. **29**: 408, 1955.

DISCUSSION

DR. G. S. CAMPBELL, Minneapolis, Minn.—Human blood has been perfused through the isolated dog lung for as long as two and one-half hours at a flow rate of 500 to 1,000 c.c. per minute.

(Slide) In order to minimize or prevent the development of pulmonary edema in the isolated lung, the following factors seem important: (1) heparinization of the dog prior to removal of its lungs; (2) open pulmonary veins to prevent back pressure (that is, the pulmonary veins are not cannulated); (3) depulsation of pulmonary artery limb of the circuit to avoid high systolic pressures in the pulmonary circulation; (4) ventilation of the lung with a pressure of 16 to 20 cm. of water, avoiding expiratory resistance; and (5) perfusion of large amounts (2,000 to 2,500 c.c.) of dextran through the lungs at a low pulmonary artery perfusion pressure (mean pressure less than 20 mm. Hg) to rid the lung of residual blood. An advantage of the "open" system as depicted here is the ease with which arterial return to the subject can be equated with venous aspiration from the subject. This balance is maintained by monitoring the level of blood in the plastic lung chamber; if this blood level falls, one knows that he is returning more blood to the subject than he is aspirating from the subject, and the opposite is true if the blood level rises.

(Slide) This shows the isolated lung. Note the plastic cannula in the main pulmonary artery, the large tube in the trachea, and the open pulmonary veins.

(Slide) Note the whitish color of the lung after it has been perfused with dextran.

(Slide) This photograph shows the lung being perfused with human blood, and I believe that the wide A-V oxygen difference is quite apparent.

Cardiac bypass has been performed in four patients utilizing this type of pump-biologic oxygenator, the longest bypass period being forty-nine minutes. The only long-term survivor among the four patients is a 123-pound boy with a traumatic I.V.S.D. The three patients who died were (1) a child with tetralogy of Fallot, severe overriding of the aorta, in whom the I.V.S.D. could not be successfully closed surgically; (2) an adult with left ventricular aneurysm and mitral disease, who died during the operative procedure; and (3) a child with I.V.S.D. with reversal of the shunt, that is, predominantly a right-to-left shunt. The method has worked well in all cases. The blood leaving the isolated lung is fully saturated with oxygen and no abnormal sequelae directly attributable to the bypass method were noted in the two short-term survivors or the one long-term survivor.

In view of the success of the method per se, we plan to use this method in more patients and in better operative risks. Following the use of the pump-biologic oxygenator, no hemorrhagic diathesis has been noted; bleeding and clotting times are normal.

DR. MELVIN M. NEWMAN, Brooklyn, N. Y.—The team working in Dr. Clarence Dennis' laboratory at the State University of New York has perfected a successful heart-lung machine which has been used experimentally for the creation and repair of atrial and ventricular defects under direct vision. The details of the apparatus were described most recently at the Society of University Surgeons' meeting at Houston in February.

(Motion picture) Here is the outflow tract of the right ventricle. The animal is on total perfusion, with the heart and lungs completely bypassed. The pulmonic valve can be seen up here and the tricuspid valve down below. Ventricular defects were made in two

ways: (1) by the cork-borer technique of Kay, Thomas, and Blalock or (2) by direct incision of the septum by a scalpel. A finger could be passed readily into the ventricle through the defect.

Blood flows in the animals were maintained at between 300 and 1,700 ml. per minute. There were 12 survivors of the 14 animals in which ventricular septal defects were produced and repaired. Some derangement of the blood-clotting mechanism occurs infrequently after perfusion and remains unexplained.

DR. J. C. CALLAGHAN, San Francisco, Calif.—In considering the problems concerned with the closure of intraventricular septal defects, I would like to tell you briefly of an approach that Dr. Gerbode and I have been testing experimentally in the laboratory at Stanford University in San Francisco. We have been impressed with the difficulties of maintaining an extracorporeal circulation, without complication, in smaller animals and we believe that in the future pump-oxygenator systems might be more difficult to manage in the infant and small child. We wish to compliment the Minneapolis group on the successful clinical application of cross circulation to this problem. In the long run, however, another simpler manner of maintaining the circulation for short periods of time must be found. To reduce this to its simplest form, we are in the process of developing a form of reservoir circulation which will function without using a pump. We have been able to perform ventriculotomy in the dry heart, using gravity alone to maintain pressure in the systemic circulation. The venous blood used in these experiments was oxygenated in specially constructed plastic bags.

(Slide) In other experiments in which we wished to have a greater outflow, we have omitted the gravity effect to see if we could, by just squeezing the plastic bag, maintain circulation adequately, and again we found that we were successful in maintaining circulation simply by using gentle compression of the transfusion bag by the human hand to develop the required pressure.

(Slide) This shows the rate of oxygenation of blood by simple agitation with an oxygen bubble in a plastic bag, showing that 85 per cent saturation is obtained from venous blood at the two-minute level and that all saturations were over 90 per cent at the end of four minutes; at the end of ten minutes all were about 93 per cent saturation. Satisfactory saturations at four minutes of agitation allow the use in the circuit of caval outflow blood collected in plastic bags containing oxygen, extending the time element after an initial four to five minutes' dependence on the reservoir blood alone. We have used this experimentally as a method of oxygenating blood.

The next slide shows an experiment in which we were able to maintain systemic pressure of 80 to 100 mm. Hg in a dog by arterial flow from a plastic bag. The upper portion of the graph shows the effect of taking pressure off the plastic bag and changing from bag No. 1 to bag No. 2. Each transfusion bag contains the amount of blood calculated at 25 to 30 c.c. per kilogram of body weight for one minute of circulation. Two bags may be emptied in a one-minute period, but this represents the upper limit of flow, approximately one liter per minute. Using low flow rates that have proved successful in clinical cases by others using controlled donor circulation, we feel that this method could be used on small children ideally and projected to children weighing up to 20 kilograms, approximately 5 to 6 years of age. We present this as a simple method of approaching this very complex problem, and as yet it is too early to evaluate.

DR. C. WALTON LILLEHEI, Minneapolis, Minn.—Direct vision for intracardiac surgical procedures improves the quality of the surgery performed, and for this reason it appears likely that the blind techniques will be progressively discarded. I am happy to see our former colleague, Dr. Cross, as well as Drs. Kay and Zimmerman, in the vanguard of the groups making this transition from blind to open intracardiac techniques.

It was just one year ago, at this meeting, that our group reported the first successful closures of ventricular septal defects. We utilized direct-vision suture closure of these defects with total bypass of the heart and lungs, maintaining the patient during this interval by means of controlled cross circulation. Physiologically, this technique has proved

to be superb; for once the circulatory systems of the patient and the donor have been linked together, and the patient has been assured of a continuous supply of oxygenated and otherwise perfectly adjusted blood from his donor. It is unlikely that a technique for total cardiac and pulmonary bypass will be developed that, for the patient's safety, excels this one. In utilizing cross circulation, however, one must be prepared to accept responsibility for the well-being of the normal, healthy donor.

(Slide) To date, 41 patients have had their heart and lungs totally bypassed for periods of from five minutes to forty minutes, utilizing the technique of controlled cross circulation. The clinical use of this method has been confined entirely to complicated septal defects, either ventricular or ventricular pathology associated with atrial defects. These 41 patients include our entire experience with this technique. There has been no donor mortality, and only one serious donor complication, previously reported, with complete recovery.

All operations have been done at normal body temperatures to obviate the crippling effects of hypothermia upon ventricular conduction. Systemic perfusion of the patient has been carried out at substantially reduced rates of flow (one-eighth to one-fourth of the normal resting cardiac output) based upon the azygos flow experimental studies. There has not been a single instance of cerebral, hepatic, or renal dysfunction attributable to these low flows. Appreciation of this low flow concept, because of the simplifications in methods made feasible, has been the most important single factor responsible for this progress. Twenty-five patients have had ventricular septal defects closed, with seven deaths. Preoperatively, all these patients have been seriously ill, and have had pulmonary hypertension. The pulmonary pressures for the entire group averaged 75 per cent of their systemic pressures. The first five patients have now been recatheterized four to nine months after corrective surgery, and in all five (100 per cent) there has been complete correction of their shunts. The pulmonary pressures in three of these five patients have already returned to normal. Numbered among these patients proved cured by recatheterization and with normal pulmonary artery pressures now is a 5-year-old girl who weighed 11 kilograms at the time of surgery and who had severe pulmonary hypertension due to three separate ventricular defects. Six of the seven deaths have been in infants, but of the 16 seriously ill infants operated upon, 10 were successfully treated. There have been no deaths due to ventricular fibrillation, complete heart block, air embolism, hemolysis, or hemorrhage.

Our next largest group subjected to direct-vision curative procedures consists of patients with Fallot's tetralogy. In this group, 10 patients, all consecutively operated upon, have had their ventricular septal defects closed and their infundibular pulmonic obstructions resected, with four deaths. All six survivors have normal color and normal femoral artery oxygen saturations, and are asymptomatic postoperatively. The first two patients have now been recatheterized, and one patient still has a small left-to-right shunt high in the right ventricle; the other boy has completely normal oxygen contents and pressures on right heart catheterization. Preoperatively, this latter boy, a desperately ill 15-month-old infant, had a femoral artery oxygen saturation of 25 per cent, which is now 95 per cent. Also numbered among the cases successfully managed by these curative techniques are a 22-month-old girl with complete atresia of the main pulmonary artery and a 10-year-old boy with tetralogy of Fallot in whom simultaneous atrial and ventricular cardiotomies were performed for correction of his defects.

As a result of these gratifying experiences, the curative operation has been adopted in our clinic as the method of choice for all patients with the tetralogy of Fallot defect who are in need of surgery at this time. For lesser degrees of disability, we recommend no surgical operation at this time but, rather, periodic re-evaluation in the clinic.

Time does not permit a discussion of the remaining types of defects successfully managed by these techniques, but I will call your attention to the fact that the atrioventricularis communis defect, contrary to some published opinions, is a correctable defect. The first patient (15 months old) with this defect has now been recatheterized postoperatively; no shunt remains and the pulmonary artery pressure has decreased from a preoperative high of 90/50 mm. Hg to 20/4 mm. Hg. This child is also normal clinically.

The value of relying upon a simple physiologic method for intracardiac surgery is attested to by the fact that the last 14 consecutive cross circulation curative operations, all carried out in seriously ill infants or children with ventricular septal defects or tetralogy of Fallot, have been performed with only one death.

Moreover, these experiences have led to the development, at our institution of two other simple techniques for total cardiac bypass based upon these same general principles, but neither of which requires a donor. Each of these two techniques has permitted successful direct-vision repair of ventricular septal defects in man. These two methods are (1) the use of a heterologous lung oxygenator, described by Dr. Campbell, and (2) the use of continuous perfusions by means of arterialized venous blood from a reservoir, to be described in tomorrow's program by Drs. Warden and Read.

In closing, I should like to emphasize that the feasibility and practicability of the direct-vision approach to intracardiac lesions is so convincing that I would venture the prophecy that coming months will see a progressive increase in its application, not only in congenital heart disease, but also for acquired heart lesions, eventually replacing blind techniques with few exceptions and making surgical management possible for conditions now quite hopeless.

DR. EARLE B. KAY, Cleveland, Ohio (Closing).—Sufficient evidence has now accumulated to demonstrate that ventricular septal defects may be a serious threat to life. With the more precise diagnostic method afforded by cardiac catheterization and the greater interest in carefully following such patients, the progressive disability noted by many such patients becomes obvious. Efforts should be directed, at the present time, to determining the exact indications and contraindications to surgical therapy, the optimum age for treatment and to perfecting surgical techniques.

There are many aspects of the problem that cannot be duplicated in the laboratory. The purpose of our paper was to present our clinical and experimental experience in the closure of such defects employing various techniques and to illustrate the manner in which the defects are most effectively closed. Undoubtedly, several methods for closure, dependent primarily upon the size of the patient, will be evolved.

With reference to Dr. Newman's motion picture, I would like to point out that the myocardiotomy is better placed if directed from the atrioventricular ring to the apex, rather than through the pulmonary outflow tract so that significant coronary vessels are not severed with resultant myocardial ischemia.

Also, as pointed out in the paper, the method shown by Dr. Cooley of placing the suture through the ventricular septum is likely to close the defect in a diagonal manner which results in incomplete closure and likely partial occlusion of the aortic outflow tract.

EXPERIENCES WITH SURGICAL REPAIR OF ATRIAL SEPTAL DEFECTS

ELTON WATKINS, JR., M.D., AND ROBERT E. GROSS, M.D.
BOSTON, MASS.

SURGICAL closure of congenital defects in the atrial septum has proved to be a useful procedure which benefits severely incapacitated patients. Our experience with the operation over a period of three years since 1952 has permitted the evolution of a number of refinements contributing to the ease of management of this condition. The refinements have been reflected in a falling morbidity rate. In the last fourteen months, mortality has been due exclusively to advanced complicated disease. The present communication describes the manner in which these changes have occurred.

PRESENT INDICATIONS FOR OPERATIVE REPAIR OF A DEFECT

Clinical and laboratory findings leading to a diagnosis of atrial septal defect have been reported previously by others.[1] Our present indications for operation were selected early in our experience when it was obvious that the procedure was risky because of unsolved technical problems. The indications were: (a) persistence of incapacitating symptoms despite optimal medical management; (b) persistence of serious cardiac enlargement and pulmonary congestion; or (c) catheterization evidence that right ventricular output was more than twice left ventricular output. By such indications patients were selected who had either severe limitation of activity or signs of impending difficulty due to enormous shunting through the diseased septum.

The use of catheter evidence of a great shunt has been strengthened by our recent experience with several adults in early middle life. These patients have tolerated enormous shunts without serious trouble through their early years, only to develop intractable congestion as cardiac reserve diminished. They have had a stormy postoperative convalescence. Improvement of exercise intolerance has not been as rapid as in the younger patients. Whenever a large atrial septal defect is suspected, cardiac catheterization should be performed to obtain precise figures as to the magnitude of the shunt, so that operative closure of the defect can be accomplished before serious congestion develops.

We have derived useful information from the calculation of systemic blood flow, pulmonary blood flow, and shunt blood flow. Although the figures are probably only rough estimates, they are accurate enough to give valuable clinical

From the Department of Surgery of the Harvard Medical School and the Surgical Service of the Children's Medical Center, Boston.

Aided by Grants from the National Heart Institute, United States Public Health Service.

Read at the Thirty-fifth Annual Meeting of the American Association for Thoracic Surgery at Atlantic City, N. J., April 24-26, 1955.

information about the volume of shunting and the existence of pulmonary vascular obstruction. Most individuals with large defects will show elevated pulmonary artery pressure despite normal pulmonary vessels because such large volumes of blood are being forced through the normal vascular bed. These patients must be differentiated from the rare individual who has actual anatomic constriction of the pulmonary arterioles. The differentiation can be made only by the calculation of pulmonary blood flow and calculation of the ratio of blood pressure to blood flow in the lungs.

We have also found it useful to correlate the volume of shunting, as calculated from catheterization figures, with the x-ray evidence of pulmonary congestion and cardiac enlargement. When the volume of shunting is large but the lungs are not seriously congested and the heart is not greatly dilated, the patient tolerates operation well because the heart is still compensating efficiently for the increased burden. In contrast, the individual with serious pulmonary engorgement and cardiac dilation, but with a shunt flow of smaller magnitude, tolerates operation poorly and develops peculiar cardiac difficulties after repair.

The presence of harsh systolic murmurs along the lower sternum has been a danger signal to us indicating the existence of either a low-lying defect (the so-called "ostium primum" defect) with associated tricuspid and mitral valve deformity or a ventricular defect with tricuspid valve insufficiency. Catheter studies cannot usually differentiate these malformations from the higher defects amenable to surgical closure.

PREOPERATIVE MANAGEMENT

These individuals present all gradations in the severity of congestive heart failure. We have not seen a patient with an uncomplicated defect whose condition could not be improved to the point of good compensation for the period of surgical intervention by such conventional measures as the limitation of fluid and sodium intake and mercurial diuresis. We have not hesitated to give digitalis in the preoperative period but only for clear-cut indications of congestive heart failure. We prefer to discontinue digitalis for several days immediately prior to operation because we have had fatal asystole in other types of patients on digitalis who had received calcium injections at the time of operation. Calcium has proved valuable in counteracting the transient myocardial weakness observed frequently during repair of the defect, and we would like to use it without the danger of synergistic action between calcium and digitalis.

Antibiotics have been given to suppress respiratory infection. We have not seen serious nutritional deficiencies. The poor body development in these patients is probably related to a deficient left ventricular output and has not been influenced by special diet. Small doses of quinidine have reduced the incidence of cardiac arrhythmias during operation. The drug has been continued after operation for several weeks.

TECHNICAL PROBLEMS AT OPERATION

Following development of the atrial well operation, it became evident that the majority of defects we saw could be closed by a simpler method of suturing from the outside surface of the intact atrium. Our technique and experience with both methods have been described in detail.[2, 3] We now plan to reserve the use of the atrial well for those malformations which cannot be closed by the external suture technique, because of either large size or anterior position in the septum. In our hands the well has proved to be a versatile method for handling such defects, but in our last

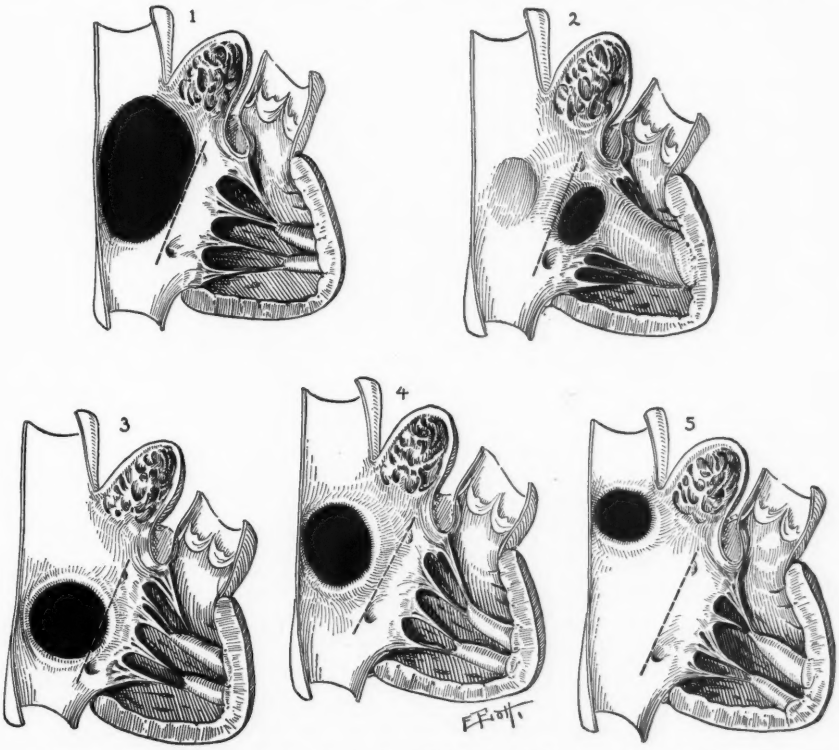


Fig. 1.—Classification of the types of atrial septal defect found in 43 operations. *1*, Common atrium (2 cases). *2*, Endocardial cushion defect (4 cases). *3*, Inferior defect (5 cases). *4*, Mid-atrial marginal defect (17 cases). *5*, Superior marginal defect (15 cases). The dotted line passing between the coronary sinus and the central fibrous body serves to locate the atrioventricular conduction system.

30 patients only 3 defects were types which would have required use of the atrial well. In one of the patients an error was made in not applying the well. Death followed attempted closure of a large defect. In the other two patients associated tricuspid regurgitation resulted in such a high atrial pressure that the well could not be applied. Commonly the defects we have seen have been located high and posterior in the atrium near the root of the right pulmonary

veins. The preponderance of dorsal defects has led us to develop a simple classification of surgical anatomy. Each class presents unique characteristics and problems in surgical management.

SURGICAL ANATOMY OF ATRIAL SEPTAL DEFECTS

Although the 43 defects evaluated at operation have varied greatly in size and shape, they tended to fall into five general categories, depending upon their location in the septum (Fig. 1). Certain orifices have been multiple or slit shaped but surgical management was similar to the repair of a solitary oval defect in the same location.

1. Common Atrium.—In this heart the septum was absent except for small ridges along part of the margin of the common atrial chamber. There was no deformity of the atrioventricular valves. This type of malformation was present in 2 out of the 43 cases.

Fig. 2.—Right atrial view of an endocardial cushion defect. The defect is located at the base of the tricuspid valve. The septal cusp of the valve is absent. Superior to the defect there is an incompletely closed *foramen ovale.*

2. Endocardial Cushion Defect (the so-called "ostium primum" defect).—This defect was located anteriorly in the atrial septum immediately above the tricuspid and mitral valves. In every defect there was an associated deformity in the valve cusps below the defect. The defect was characteristically small, measuring from 1.5 to 2.5 cm. in diameter. It was frequently separated from the upper atrial septum by a prominent ridge of tissue so that the orifice entered

into a small vestibular chamber isolated from the main cavity of the right atrium. Above this diminutive antechamber, a foramen ovale was found, frequently bearing a second defect (Fig. 2).

We prefer to classify this defect as one of the endocardial cushion because it is related not only to inadequate fusion of the primitive atrial septum with the endocardial cushions (the anlage of the mitral and tricuspid valve rings) but also the incorrect maturation of the endocardial cushions themselves. This

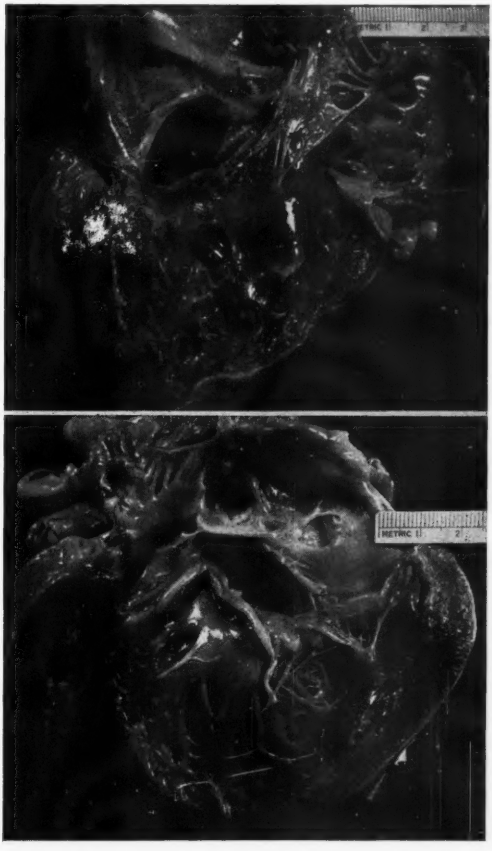


Fig. 3.—Right atrial view (top) and left atrial view of an endocardial cushion defect. The septal cusp of the tricuspid valve is fenestrated. There is a V-shaped defect with wide incompetency of the mitral valve.

maldevelopment results in serious atrioventricular valve deformity and regurgitation. We have seen this defect 4 times in the 43 cases. Each time it has been associated with deformities of both the mitral and tricuspid valves. Although we have been able to close some of these defects surgically, cardiac failure and death have promptly supervened. We question the wisdom of at-

tempting to convert the left atrium into a small closed chamber into which blood regurgitates without any passageway for decompression through the surgically closed defect.

Dissection studies of the atrioventricular conduction bundle indicate that this specialized tissue makes up the inferior margin of the endocardial cushion defect. Animals have commonly shown serious conduction disturbances and death when this bundle was traumatized with needles or cut.

The endocardial cushion defect should be differentiated at the time of operation from the following type of defect, the inferior defect, which it closely resembles. The inferior defect presents an entirely different picture in the ease and value of surgical closure.

3. Inferior Defect.—The inferior type of defect was located close to the inferior caval orifice. The right atrial chamber is shaped like an inverted cone with a broad superior surface bearing the superior caval orifice and the right atrial appendage. Inferiorly the caval orifice extends through the full diameter of the narrow chamber. Consequently, defects low in the septum near the inferior caval orifice extended through the full diameter of the chamber and lay in close juxtaposition to the tricuspid valve ring and coronary sinus. They resembled the endocardial cushion defects in position, but there were certain important differences: (a) the atrioventricular valves were fully developed and valvular incompetence was not apparent; (b) the defects were larger than the endocardial cushion defects, ranging in diameter up to 4.0 cm.; (c) there was no evidence of a *foramen ovale* or *fossa ovalis* palpable superior to the defect; (d) there was no evidence of tissue ridges or antechamber formation superior to the defect.

The adequate development of the atrioventricular valves is of considerable significance to the surgeon since there is no valvular regurgitation which might contraindicate the conversion of the left atrium into a high-pressure chamber without outlet through the defect. The well-developed annular rings form a broad shelf suitable for the placement of secure sutures.

As the surgeon palpates these defects of the inferior type, certain features allow differentiation from the endocardial cushion defect: (a) large size; (b) absence of regurgitant jets at the mitral and tricuspid orifices; (c) a smooth residual septal surface above the defect without fossa ovalis or prominent ridges. The absence of a separate fossa ovalis suggests that this defect results from imperfect development of the *septum secundum* during embryonic life.

The inferior defect does resemble the endocardial cushion defect in its close proximity to conduction tissue in the tricuspid valve ridge. We have been able to close these defects with a feeling of safety only after certain landmarks were found which allow the surgeon to guide sutures away from the node tissue.

We have seen the inferior type of defect 5 times in our 43 cases. Hence it tends to be about as common as the endocardial cushion defect.

4. Mid-atrial Marginal Defect.—The commonest type of defect we encountered was one located near the posterior margin of the atrial septum adjacent to the roots of the right pulmonary veins about midway between the two caval orifices. It was present 28 times in our 43 cases. The close

proximity to the dorsal margin of the atrium and the position well away from the caval orifices render these defects particularly suited to closure by the external suture techniques. Difficulties in surgical closure stem from the large size of the defect (up to 4.0 cm. in diameter) and the thin friable character of the anterior remnant of septal tissue.

5. Superior Marginal Defect.—Next to the mid-atrial marginal defect, this deformity was the most common. It has been present in 15 of our 43 cases. The lesion tended to be smaller than the mid-atrial marginal defect and lay in close proximity to the superior caval orifice. External suture closure must be made in such a manner that occlusion of the superior cava is avoided. We have seen no arrythmias suggesting injury to the sinoauricular node during closure of these defects and have not carried out anatomic studies locating the structure.

Major associated conditions have been divided sharply among the types of defects. True pulmonary vascular obstruction has been seen only with the mid-atrial marginal type of defect. Possibly the large size of this malformation results in tremendous blood shunting early in life thus predisposing to pulmonary vascular obstruction. Anomalous drainage of the right pulmonary veins into the right atrium has been seen only with defects of the inferior and superior marginal types. The eccentric position of the defect in relation to the mid-atrial location of the anomalous vein orifices presents some difficulty in surgical management. Deformities of the mitral and tricuspid valves have been present only in defects of the endocardial cushion type. The inferior type of defect differs from the endocardial cushion defect in the absence of valve deformity, but resembles it in the proximity to the valves and atrioventricular nodal tissue.

Lesions suitable for closure by the external suture method have been the inferior, mid-atrial marginal, and superior marginal defects. The endocardial cushion defect may prove suitable for closure by external suture but the associated valve deformity introduces serious problems after closure. The common atrium type of defect would seem to require some type of prosthesis or tissue graft to avoid atrial and valve-ring distortion. The occurrence of the inferior type of defect, which is suitable for closure by palpatory methods, indicated the necessity for dissection studies to locate the atrioventricular node and bundle, and to determine whether or not landmarks could be found to enable the surgeon to avoid the structures during external suture closure of the inferior defect.

SURGICAL ANATOMY OF THE ATRIOVENTRICULAR CONDUCTION SYSTEM

A series of normal hearts and hearts bearing atrial septal defects were dissected by the technique described by Widran and Lev.[4] This series included the heart of a patient who had died during the course of an apparently uncomplicated closure of a defect when a suture had been passed about a major left bundle branch of the conduction system. The rapidly deteriorating course of this patient immediately following the onset of nodal rhythm on tying of the suture further indicated the hazard of node injury.

In the normal heart (Fig. 4), the main atrioventricular node, the main bundle, and all bundle branches lie below a line connecting the Thebesian valve to the central fibrous body in the tricuspid annulus. Septal defects of the types in which the orifice lay well above this line (superior and mid-atrial marginal defects) presented a considerable remnant of septum containing no specialized conduction tissue. Defects of the types in which the orifice lay along or anterior to this line (inferior, common atrium, and endocardial

Fig. 4.—Dissection study of the atrioventricular conduction system in a normal heart of a newborn infant. The tricuspid valve (*TV*) has been partially excised and the atrioventricular node (*N*) exposed beneath the right atrial endocardium. The main bundle passes to the right from the node to bifurcate into left bundle branches (divided) and the right bundle branch which passes downward to the right ventricle (*RV*). A dotted line passing from the coronary sinus (*CS*) to the central fibrous body (*CFB*) forms a landmark line above which conduction tissue is not found. The coronary sinus and central fibrous body may be palpated at the time of operation. A probe-patent *foramen ovale* (*FO*) is present.

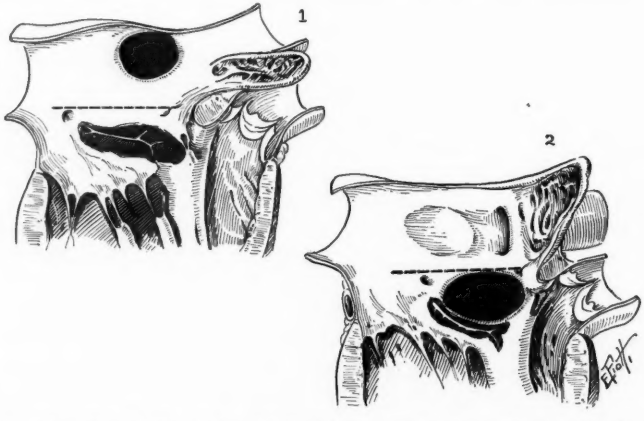


Fig. 5.—Dissection study of the atrioventricular conduction system in hearts with atrial septal defects. *1*, With a defect of the superior marginal type (Type 5), the conduction system is located well away from the lower margin of the defect. The line connecting the landmarks of the coronary sinus and central fibrous body indicates the most superior limit of any conduction tissue. *2*, With a defect of the endocardial cushion type (Type 2) the entire orifice of the defect is located below the line. In such a defect the atrioventricular node makes up the inferior margin of the orifice.

cushion defects) all had conduction tissue in the anterior edge of the defect. Definition of this line by palpation at operation should permit evaluation of the position of the specialized conduction tissue.

It was found that both the Thebesian valve and the central fibrous body could be palpated easily by the exploring finger placed in the atrial appendage at the time of defect closure. The coronary sinus orifice is located at the anterior edge of the inferior vena caval orifice. The Thebesian valve feels like a taut, paper-thin ridge over this orifice. When the finger is swept upward from the Thebesian valve along the septal segment of the tricuspid valve annulus, it comes to rest naturally in the commissure between the septal cusp and the infundibular cusp of the tricuspid valve. At that point the central fibrous body presents on the surface of the tricuspid annulus as a pitted depression 2 to 3 mm. in diameter. Location of these two points permits orientation of the position of the conduction system, which lies anterior to the line. The atrioventricular node and entire bundle are located at the margin of the endocardial cushion type of defect. The inferior type of defect, on the other hand, may present only a small segment of rim close to the node and adjacent segment of bundle. The remainder of the bundle and its branches will lie anteriorly and superiorly deep in the well-formed superior remnant of atrial septum. The anterior portions of the bundle and branches are more variable in location than the node and adjacent segment of bundle, so persistence of the superior septal remnant greatly simplifies surgical management. Definition of the landmarks and connecting line is now used routinely during the exploration of all atrial septal defects.

SPECIAL TECHNICAL PROBLEMS IN CLOSURE OF DEFECT BY THE EXTERNAL SUTURE METHOD

Of the five groups of atrial septal defects, the three groups located posteriorly in the septum (inferior, mid-atrial marginal, and superior marginal) were best suited for closure by the external suture method. Since these three groups make up 86 per cent of the defects in our experience, the external suture method has been most commonly used. Each of the three groups presents special problems in closure.

Inferior Defects.—In the closure of this type of defect, we have made use of the method of defining the position of the atrioventricular node. The tricuspid and mitral valve annuli are well formed and create a broad shelf suitable for placement of sutures. Once the position of the A-V node is defined, sutures can be passed into the left atrial side of the shelf, more on the mitral annulus than the tricuspid annulus. This avoids the conduction system which lies beneath the endocardium on the right atrial side of the shelf.

After extensive mobilization of the left atrium inferior to the right pulmonary veins, sutures may be introduced into the left atrium inferiorly near the anterior lip of the defect. This maneuver has the advantages of eliminating the possibility of inferior caval obstruction, of placing the least tension on the suture line, and of creating a suitable arc for passage of the needle outward after it has traversed the anterior lip of the defect. After placement of

the most inferior sutures in this manner, the succeeding sutures pass more to the right of the right inferior pulmonary vein, thereby creating a suture line which curves upward around the vein. Neither the vein nor inferior vena cava is obstructed. The steps in this method of repair are shown in Figs. 6 and 7.

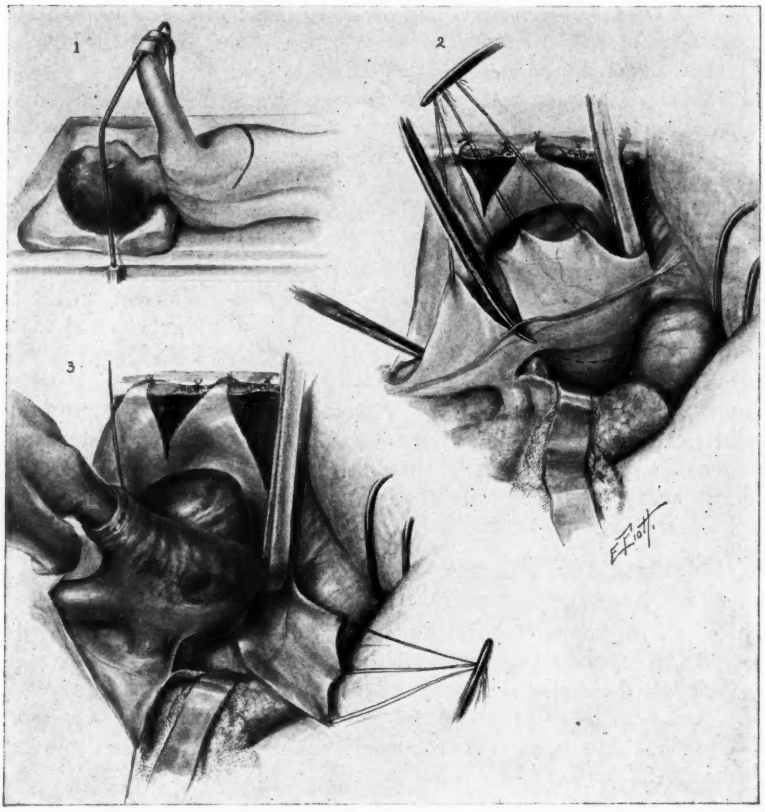


Fig. 6.—External suture closure of an atrial septal defect of the inferior type. *1*, Position of the patient on the operating table. *2*, Following exposure of the heart, the auricle is explored through the atrial appendage to evaluate the type of defect present. If a Type 3 defect is present, the right phrenic nerve is mobilized and the pulmonary ligament and pericardium are divided posteriorly. *3*, Retraction of the lung and posterior pericardial flap provides exposure of the posteroinferior aspect of the left atrium near the defect.

Mid-atrial Marginal Defects.—The septal remnant anterior to this large defect may be quite attenuated. Care must be taken to avoid tearing of sutures. In large defects we place all sutures before tying so that all sutures may be tightened to distribute tension evenly. A useful instrument has been designed to push the atrial wall downward to meet the septum in those cases where the right atrium is greatly dilated with blood. If the other digits of the palpating hand are passed behind the atrial appendage into the groove occupied by the aorta, the atrial septum may be pushed posteriorly, separating it from the ascending aorta and relieving tension as sutures are tied.

On many occasions we have made use of the maneuver described by Bailey,[5] of invaginating the lateral right atrial wall into the defect. Defects of the mid-atrial marginal type are suitable for such invagination since they lie at a considerable distance from either caval orifice and the cavae are not compromised by invagination. We have attempted, however, to secure the invaginated right atrial wall to all edges of the defect in order to create a closure which is flush with the septal surface of the left atrium. This avoids the creation of a diverticulum containing suture material and traumatized tissue which might act as a source of emboli to the systemic circulation.

Superior Marginal Defects.—These small defects have been the easiest to repair. On occasion it has been useful to mobilize the superior vena cava and

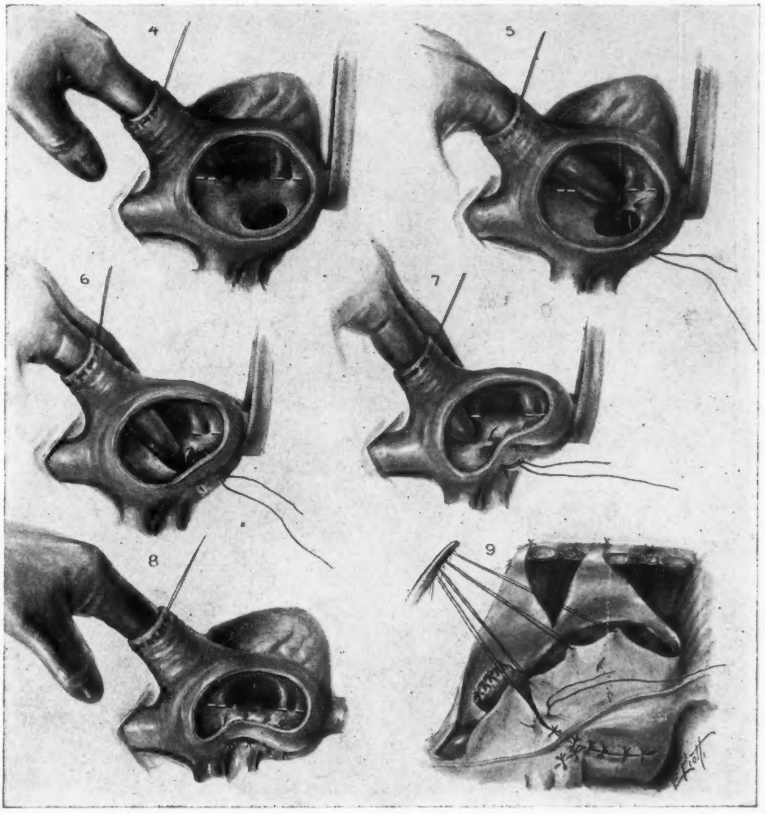


Fig. 7.—External suture closure of an atrial septal defect of the inferior type. *4*, By palpation, the line connecting the coronary sinus and central fibrous body is defined. Atrioventricular node tissue lies on the right ventricular side of this line. Although the inferior type defect abuts on the valve rings, there is a broad shelf of mitral valve ring inferiorly which contains no conduction tissue. *5*, The first suture is passed into the left atrial wall which lies close to the anterior lip of the defect. *6*, The suture passes through the mitral shelf and emerges from the heart through the wall of the right atrium. *7*, Successive sutures pass to the right side of the right inferior pulmonary vein. *8*, Closure is thus effected by a suture line which curves around the orifice of the inferior pulmonary vein. *9*. Repair of the pericardial defect and closure of the atrial appendage.

retract it upward with an encircling tape so that sutures may be placed toward the left atrial side of the right pulmonary veins in a manner similar to the closure of the inferior type of defect. The method of closure is shown in Fig. 8.

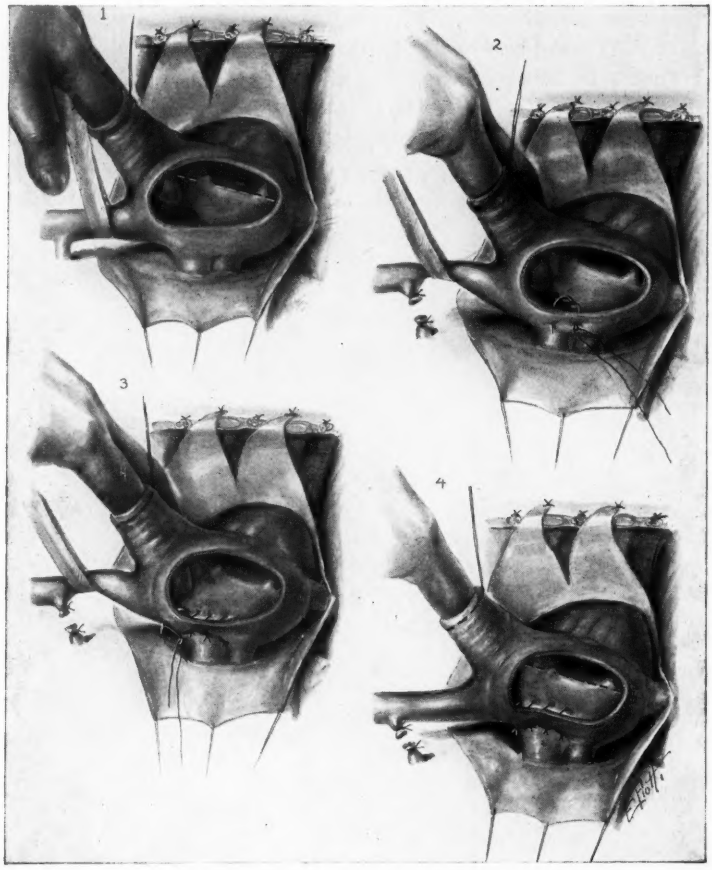


Fig. 8.—External suture closure of an atrial septal defect of the superior marginal type. *1*, The usual pericardial opening is extended by complete mobilization of the superior vena cava from the pericardial reflexion. The defect is explored and the position of the atrioventricular node defined. A traction tape is placed about the superior vena cava. *2*, Inferior sutures are placed to the right of the root of the pulmonary vein. A finger passed behind the atrial appendage into the aortic groove pushes the septum posteriorly to compress the orifice of the defect and push it away from the ascending aorta which lies behind the right atrium. Retraction of the superior vena cava compresses the orifice of the defect and facilitates the placement of sutures. *3*, Superior sutures are passed into the heart through the left atrial wall to avoid occlusion of the superior caval orifice. *4*, The defect is closed by a suture line which curves about the orifice of the superior pulmonary vein.

ANOMALOUS PULMONARY VENOUS DRAINAGE WITH ECCENTRIC DEFECT

Since anomalous drainage of the right pulmonary veins into the right atrium has been associated exclusively in our experience with inferior or superior marginal defects, the veins have drained into the atrium some distance in-

ferior or superior to the orifice of the defect. We have found it useful to insert a small tonsillectomy knife through a pure-string suture in the wall of the right atrium. Under control of the palpating finger, this knife is used to enlarge the atrial defect upward or downward so that the enlarged orifice may be sutured to the right of the transposed veins. This maneuver corrects the anomalous venous drainage and obliterates the interatrial communication at the same time. It is useful to define the landmarks of the conduction system in planning the location of this incision so that the enlarged defect and following sutures can be located away from the node.

POSTOPERATIVE MANAGEMENT

Patients have presented few special problems in postoperative care. Each patient receives quinidine. An indwelling catheter is used to measure urinary output accurately at hourly intervals. The measurement is useful during the first few postoperative days in determining whether or not the patient is retaining fluid. If this is the case, it may be anticipated that the patient will develop a peculiar syndrome of transient left ventricular failure. The failure is presaged by the retention of fluid and gain of weight during the first few days after operation. Between three and fourteen days after operation, such patients have complained of pain and tenderness in the right upper quadrant. Definite signs of hepatic enlargement then appear. X-ray studies at that time have shown pronounced enlargement of the left atrium and progression of the pulmonary congestion which was present before operation.

The syndrome must be recognized promptly since it is accompanied by electrolyte derangements and hemodilution due to retention of fluid. The falling hematocrit must not be interpreted as an indication for blood transfusion. Transfusions will increase the blood volume further and aggravate the condition. Plasma volume studies indicate the true picture—a constant red cell mass and rising plasma volume with resultant reduction of peripheral hematocrit.

Arrythmias are frequent during this period. The most common disturbance is auricular flutter. Conventional methods of medical therapy have been employed but the most valuable therapy in management of the arrhythmia has been directed toward control of the underlying congestive failure. The arrythmias tend to disappear spontaneously once compensation is achieved.

Our difficulties became less frequent once we began to shape our immediate postoperative care in anticipation of a period of transient failure. Fluid intake has been limited in all patients. The afrebrile adult receives a maximum of 1,500 c.c. per day. Sodium intake has been restricted. Blood replacement has been gauged by loss measured at operation and through thoracotomy tubes. We have used mercurial diuretics without complications. We have not restricted the use of blood transfusion during operation or during the immediate postoperative period, but have routinely given 20 to 50 c.c. of 50 per cent glucose intravenously immediately after operation. We have frequently observed mild hypotension during the immediate recovery period.

When this is unaccompanied by oliguria and signs of peripheral vascular collapse, we are not very much concerned, but serious hypotension accompanied by oliguria has been treated by transfusion. Digitalis has been given if signs of congestive failure are seen. When the requirements for blood replacement appear greatly in excess of operative blood loss and chest tube drainage, vasopressors have been valuable adjuncts to transfusion.

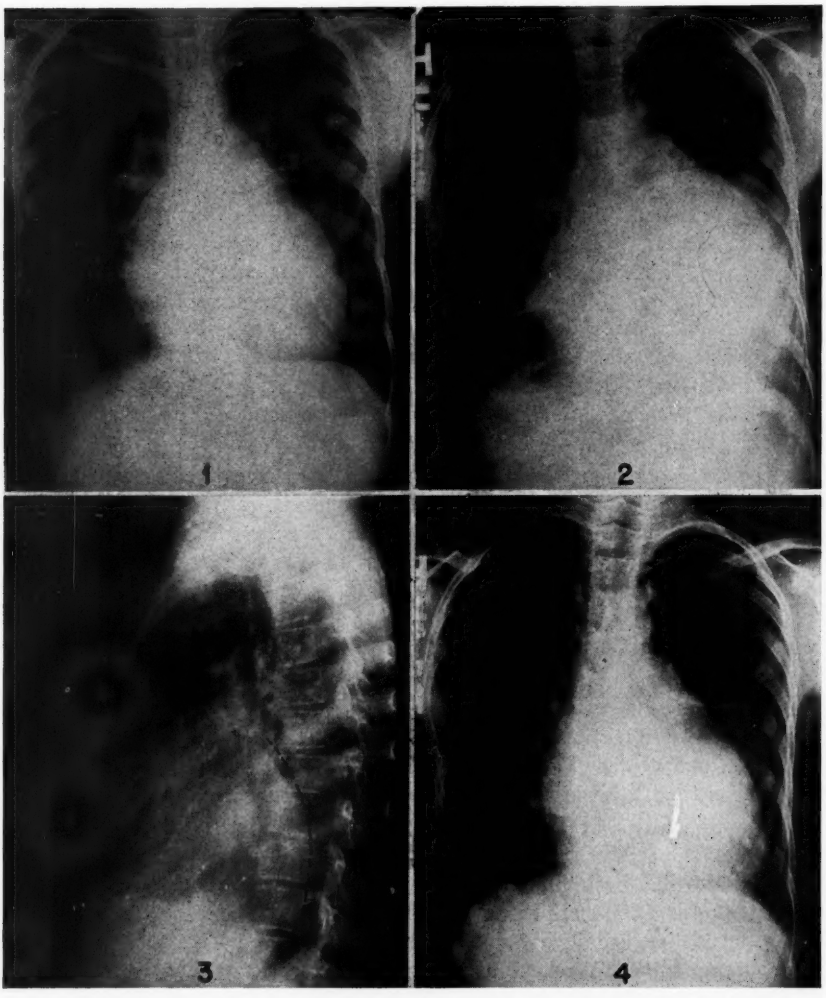


Fig. 9.—Chest x-rays of a child showing transient left ventricular failure after atrial septal defect closure. *1*, Preoperative film. The heart is enlarged and there is pulmonary congestion. *2*, Nine days after closure of a large defect. There is failure of the left ventricle with a large heart and increased pulmonary congestion. *3*, Left lateral view showing left atrial enlargement. *4*, Seven weeks after operation there is decrease in right atrial size and decreased pulmonary congestion.

Studies by our associates suggest that the transient left ventricular failure may be related to the altered conditions under which the left ventricle is functioning after operative closure of the atrial defect.[6] Preoperative studies indicate that an atrial septal defect impairs filling of the left ventricle by acting as a vent through which blood is shunted to the low-pressure venous system. Since the left side of the heart is unable to maintain an adequate diastolic filling pressure, left ventricular stroke output is diminished and the

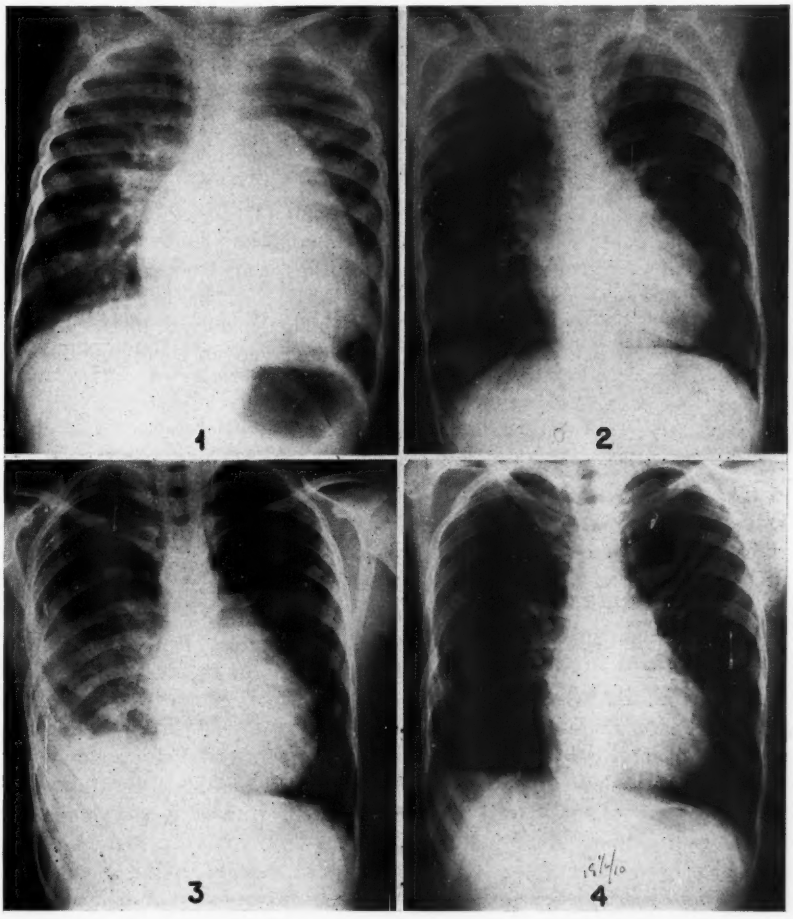


Fig. 10.—Chest x-rays of children with atrial septal defects and after operative repair. *1*, A 4-year-old child presented serious symptoms and great cardiac enlargement with only a small shunt flow (1.5:1 ratio of pulmonary flow to systemic flow). This combination of findings suggests that the course will be poor. The patient died of congestive failure after operation. *2*, In contrast, a 7-year-old girl presented enormous shunting (4:1 ratio of pulmonary flow to systemic flow) with only minimal signs of cardiac enlargement and pulmonary congestion. This combination of findings suggests a benign course. *3*, Four days after operation there is no evidence of left ventricular failure. The course was uncomplicated. *4*, Seven months after operation the heart is small and the lung fields uncongested.

ventricle remains small at all times. It seems logical to assume that the diminution of left ventricular volume over the period of years prior to closure of the defect would impair the growth of the left ventricle and impose an unaccustomed burden upon it immediately after the defect is closed at operation. We have had occasion to see several autopsy specimens in which the left ventricle was diminutive in the presence of a large septal defect. The mitral valve was hypoplastic and a pathologic diagnosis of "mitral stenosis" had been rendered although the valvular deformity was obviously not of rheumatic origin.

Postoperative Embolization.—Disconcerting neurological changes have been seen in 3 patients. Presumably thrombotic emboli had broken away from the left atrial surface of the defect repair. In 2 patients, transient minor changes were gradually resolved. In an 8-year-old girl, coma and decerebrate changes occurred suddenly twenty-four hours after an uncomplicated external suture repair of a mid-atrial marginal defect closed by a U-shaped suture line. This closure may have left an appreciable diverticulum of excluded right atrial chamber in communication with the left atrium. Death followed the development of serious electrolyte disturbances. Autopsy could not be obtained.

We can prove no definite cause for embolization, but believe that minimization of tissue trauma and avoidance of appreciable depressions in the left atrial surface of the defect closure will reduce the incidence of such complications. Pulmonary embolization has not been seen.

RESULTS OF OPERATION

We have performed exploratory operations on 46 patients with a preoperative diagnosis of atrial septal defect. In 3 patients, the diagnosis of atrial spetal defect was in error despite clear-cut signs and adequate preoperative catheterization studies. The common cause of this error was a ventricular septal defect with tricuspid regurgitation which could not be differentiated from an uncomplicated atrial septal defect by preoperative studies.

The remaining 43 patients had an atrial septal defect. In the first consecutive group of 22 patients, there were 9 deaths. Six of the deaths were due to technical errors which have proved avoidable with increasing experience. In the last 21 patients, there have been 4 deaths. Two deaths followed attempted closure of endocardial cushion defects with pronounced valvular deformity. Two deaths were due to immediate congestive heart failure following uncomplicated closure of mid-atrial marginal defects with advanced pulmonary vascular obstruction. None of the procedures were complicated by avoidable technical errors. Consequently, we now regard defects with associated anatomic valve deformity or with advanced pulmonary vascular obstruction as not amenable to repair by our present methods.

The diagnosis of true pulmonary vascular obstruction can be made by adequate preoperative catheterization studies. True pulmonary vascular obstruction must be differentiated from the common innocuous pulmonary hypertension due to tremendous flow through a normal pulmonary vascular bed.

Differentiation can be made only by calculation of pulmonary arteriolar resistance or some similar index based upon the ratio of pulmonary mean arterial pressure to pulmonary blood flow. Normal values for these indices have been published.

We do not know of any way short of digital exploration of the atrium to diagnose the endocardial cushion type of defect with valve deformity. Its presence may be suspected in the patient who presents lower sternal thrills and harsh systolic murmurs in the absence of catheterization evidence of an associated ventricular septal defect.

We have not performed postoperative cardiac catheterization studies during the time of the hospitalization for operation because we believe that the patients should be restored to full activity prior to evaluation of the results of operation. In one patient this policy has been supported by the reappearance of a systolic pulmonic murmur some time after discharge, suggesting reopening of a small shunt. A small shunt was present at the time of late postoperative catheterization.

Nine patients have been catheterized. Six children have been studied. Complete closure of the defect has been demonstrated in four. In 2 of our earlier cases, there is evidence of persistence of a greatly diminished shunt. It is also encouraging to see a pronounced difference between right atrial pressure and left atrial pressure after operation in all patients with closed defects. The rise of left atrial pressure ("pulmonary capillary" pressure) with the reduction of heart size and pulmonary congestion indicates that closure of a defect allows the left atrium to fill more effectively under a higher pressure. We hope that this will allow the left ventricle to fill more efficiently and to develop normally after operation.

The success of closure of defects in the adults cannot be evaluated from our present data because we have selected chiefly those patients suspected of residual shunting for early catheterization. Of 4 such patients, the shunts have been only slightly reduced in 2, reduced to one-third in one, and completely obliterated in one. The last patient has shown a rise of 9 mm. in pulmonary capillary pressure.

Clinical improvement has been striking. All patients have reported an increased exercise tolerance and relief of symptoms. Many of the younger patients with large shunts were reported as asymptomatic prior to operation but the degree of their incapacity became obvious to the parents only after surgical repair allowed the children to compete efficiently with their schoolmates at play. Convalescence in the adult patients has been slower than in the children.

The characteristic murmurs of atrial septal defect have altered after operation, but persistence of some type of pulmonic murmur has been seen in several children whose defects have been proved to be repaired by catheterization. The murmur may be related to anatomic deformity of the large pulmonary vessels since it tends to diminish gradually with growth of the patient. The persistence of apical diastolic murmurs has been a more reliable sign indicat-

ing the persistence of appreciable shunts after operation. This type of murmur has disappeared in patients in whom complete closure of the defect has been accomplished.

SUMMARY

1. Surgical repair of atrial septal defects has proved to be a useful procedure in our hands.

2. Arbitrary indications for operation were selected early in our experience to include only those patients with considerable evidence of disability or impending difficulty from large shunts. The indications we have used are: (a) persistence of incapacitating symptoms despite optimal medical management; (b) persistence of serious cardiac enlargement and pulmonary congestion, or; (c) catheterization evidence that right ventricular output was more than twice left ventricular output.

3. The common location of defects along the posterior margin of the atrial septum has made it possible to utilize external suture methods exclusively during management of our last thirty patients. The atrial well method is more versatile but is not necessary for the repair of most defects.

4. A classification is made of the surgical anatomy of forty-three defects observed at operation. This classification has been made with the intention of differentiating those low-lying defects associated with atrioventricular valve deformity from defects without valve deformity lying near the inferior vena cava. Serious regurgitation with the former type of defect has been aggravated by surgical repair. The latter type of defect is amenable to surgical repair but presents technical difficulties in the danger of injury to specialized conduction tissue in the atrioventricular node and bundle. This tissue may be located by palpation of two landmarks within the atrial chamber—the valve of the coronary sinus and the central fibrous body. Conduction tissue is not found posterior to a line connecting these two landmarks.

5. When a defect lies near a caval orifice, it is useful to employ sutures introduced into the heart through the left atrial wall. This avoids constriction of the caval orifices by suture.

6. Postoperative management of patients has been complicated on occasion by the appearance of transitory left ventricular failure. All patients are placed on a prophylactic regime of fluid limitation during the immediate postoperative period. Digitalis and mercurial diuretics are given if failure develops. Arrythmias have been common in patients during this period of failure.

7. Catheterization evidence of the success of closure of the defect is not complete. Evidence of a rise of "pulmonary capillary" pressure after successful closure of the defect indicates that operation improves the diastolic filling of the left ventricle.

8. During our last twenty-one consecutive cases, deaths have occurred only in the presence of advanced disease—advanced pulmonary vascular

obstruction in two patients and serious atrioventricular valve deformity in two patients. We now regard these conditions as contraindications to defect closure by our methods.

REFERENCES

1. Braudo, J. L., Nadas, A. S., Rudolph, A. M., and Neuhauser, E. B. D.: Atrial Septal Defects in Children: A Clinical Study With Special Emphasis on Indications for Operative Repair, J. Pediat. 14: 618-631, 1954.
2. Gross, R. E., Watkins, E., Jr., Pomeranz, A. A., and Goldsmith, E. I.: A Method for Surgical Closure of Interauricular Septal Defects, Surg., Gynec. & Obst. 96: 1-23, 1953.
3. Gross, R. E., and Watkins, E., Jr.: Surgical Closure of Atrial Septal Defects, Arch. Surg. 67: 670-681, 1953.
4. Widran, J., and Lev, M.: The Dissection of the Atrioventricular Node, Bundle and Bundle Branches in the Human Heart, Circulation 4: 863-867, 1951.
5. Bailey, C. P., Bolton, H. E., Jamison, W. L., and Neptune, W. B.: Atrio-septo-pexy for Interatrial Septal Defects, J. THORACIC SURG. 26: 184-219, 1953.
6. Hickler, R. B., and Goodale, W. T.: Atrial Septal Defect: Relation of Interatrial Pressure Gradient to Right and Left Ventricular Work in the Effects of Surgical Closure, Federation Proc. 13: 70, 1954.

DISCUSSION

DR. CHARLES P. BAILEY, Philadelphia, Pa.—There is really not much to be added to this presentation of Dr. Gross and Dr. Watkins. We performed the first atrioseptopexy operation in 1952, in February, and since that time have operated on 73 more patients, on whom this technique or some modification was used. Among them were 57 with so-called septum secundum defects. There were 5 deaths in that group. With 3 exceptions those who have been recatheterized—about two-thirds of the entire series— have been proved to have complete closure of the defect. We operated on 17 primum defects and we have a mortality there that I am ashamed of—more than 60 per cent. Therefore, we have stopped operating on septum primum cases. We have used the supine position rather than the lateral position which Dr. Gross has used, and have found it quite satisfactory. Six of these patients presented coexisting mitral stenosis and this lesion was relieved at the same time; 3 of these lesions were found inadvertently during the performance of mitral commissurotomy from the right side. This approach, of course, makes it convenient to repair the defect at the same time. In 16 cases there was anomalous pulmonary venous drainage, in all but 2 to the right lung. One of these was limited to the left side and we did not do anything about it. The other was a patient with complete anomalous venous drainage, whom we operated on in two different stages. One patient had anomalous right venous drainage and no septal defect. You saw the method Dr. Gross used for diverting the anomalous right pulmonary venous drainage into the left atrium, and of course we used practically the same method. But in this instance there was no septal defect. After due consideration we created a posterior septal defect and then sewed the right atrial wall to the anterior margin of the created defect, and in that fashion we rerouted the venous blood from the right lung into the left atrium.

I cannot congratulate Dr. Gross enough for his efforts to demonstrate exactly where the common conduction bundle lies. That is a very important landmark. Up to now we have been guided solely by the location of the coronary sinus ostium and have tried to keep our suture line above the valve. Unfortunately, in 4 of our 16 primum cases we did interrupt the conduction bundle. Since 3 of these 4 patients died, we thought it was a most serious accident in contrast to certain other more favorable reports of recovery after surgical division of the conduction system.

We always mobilize the atrial wall if it does not seem large enough for our purposes by the method Bahnson has suggested; that is, a separation of the interatrial groove. If one puts a finger in the right atrium so that one does not separate the groove all the way up to the defect, the additional separation is not hazardous. When sufficient atrial wall has been mobilized, one can readily repair, I find, even a defect which is more common with us than

the posterior one just described; that is, one just behind the aorta running up as high as the superior vena cava. There is a posterior septal margin, but none anteriorly. Perhaps we see this anterior defect more commonly because most of our patients are adults.

DR. DENTON A. COOLEY, Houston, Texas.—The excellent results presented by Drs. Watkins and Gross using mostly closed methods of repair of atrial septal defects are very impressive. I too am convinced that a large number of such anomalies can be corrected without resorting to temporary interruption of the circulation. Some problems in cardiac surgery inevitably will be solved by technical improvements alone. It is true that open heart surgery is developing rapidly with its many obvious advantages. On the other hand there is a certain inherent risk involved in temporary by-pass of the heart, and this may be especially important in patients with severe cardiac embarrassment. Such risks could be avoided by use of a simplified method based largely upon anatomic features of atrial defects which facilitate their repair.

Although my experience with surgical closure of interatrial septal defects is comparatively small, the results have been encouraging. In my opinion the modifications devised by several Scandinavian authors of Dr. Gordon Murray's technique of septal suturing provides the simplest and most effective method of dealing with septum secundum and some ostium primum defects. An advantage of the purse-string suture is that it will close multiple defects when they exist. Addition of a couple of mattress sutures through the septum reinforces the closure and prevents the purse-string suture from pulling through the atrial wall.

I have tried a similar technique of suturing for ventricular septal defects in which mattress sutures are passed through the ventricular septum. This method was tried in 3 patients and one died from hemorrhage twenty-four hours later when the mattress suture pulled through the myocardium. In the 2 surviving patients there were no complications and the defects appear to be reduced significantly in size. Although these results are not as impressive as some reported by Lillehei where cross-circulation was used, it may be that the procedure will have some usefulness in infants and small children. One should be ready to combine the best features of all methods which have been proposed, if necessary, in the development of an ideal operative technique.

DR. CONRAD LAM, Detroit, Mich.—I should like to present a modification of the technique of atrioseptopexy. This method embodies some of the features of the methods used by Dr. Bailey and by Dr. Gross.

(Slide) This autopsy specimen illustrates the anatomy of interatrial septal defect. One notes the large defect, the tricuspid valve, and the orifices of the venae cavae. If the posterior rim of the defect is pushed anteriorly, the defect is closed. If it is maintained in this position by sutures, a satisfactory repair has been obtained. These sutures can be placed by a closed technique, with the use of a two-pointed needle with the silk swaged on in the middle.

(Slide) We first carry out the Sondergaard dissection, separating the pulmonary veins from the right auricle. This permits the needle to be inserted either through the posterior rim or just to the left of it. The needle then passes through the anterior rim of the defect, and the point emerges from the auricular wall at any convenient place. It is grasped with the needle holder and when the trailing point has cleared the anterior rim, the direction of the needle is changed so that the trailing point emerges from the auricle near where the needle first entered.

(Slide) As many sutures as necessary are placed to close the defect completely. There is no danger of interfering with vena caval flow or the conduction mechanism.

We have closed 13 defects of the septum secundum type by the closed method, without a death. We encountered 4 of the so-called ostium primum type. These could not be closed by the external method and 2 patients succumbed after exploration.

The two-pointed needle is similar in design to that which Dr. R. A. Cowley has used for blood vessel anastomosis.

DR. HENRY T. BAHNSON, Baltimore, Md.—We have encountered a fair number of anomalies of the venous return in extremely ill pediatric patients with auricular septal defects. Most present no problem as they can be closed by shifting the septum as Gross and others have described.

(Slide) In patients such as the one shown here the problem is a different one. This patient had a total anomaly of the pulmonary venous return, all the veins draining into an additional atrial chamber. We debated quite a while trying to decide what to do, and ultimately came up with an idea that may be helpful to some of you. We had previously dissected in the groove between the atrium and the pulmonary veins, with the technique Dr. Bailey ascribed to me but which goes back to Sondergaard as others have mentioned. This is very helpful in anomalous venous return because if one dissects in this groove he cannot miss the anomalous drainage. We simply began with a purse-string suture adjacent to the aorta as Crafoord and Bjork have described, continued this on around and took bites on the anterior wall of the right atrium; the right atrium in this condition is large and quite roomy. This is tied down, and as you see we have converted a heart with three atria into one with four atria. We have made the inferior vena cava and the coronary sinus vein drain through the tricuspid valve. The pulmonary venous drainage which came into the third atrium went through a septal defect to the remaining right and thence to the left atrium. In essence this left the superior cava draining with the pulmonary veins to the left heart, so we tied down the superior cava between the azygos vein and the right auricle over a nerve hook to obstruct this flow and direct it through collaterals to the inferior cava. We later wrapped this with a piece of cellophane. We were quite gratified to find that when the pressures were measured—we measured pressures prior to placing the suture, and following—the pressures in the pulmonary vein, the original right atrium, our new right atrium, and the third atrium, all had fallen from the preoperative values. The pressure in the superior vena cava, the drainage of which was partly constricted, was similar to that which had been recorded before the procedure. The patient made an essentially normal recovery except that we noticed for the first few days he did not like to lie flat, a common symptom of the superior caval syndrome. At the time of his discharge he was lying flat.

This is the condition which I think is present in many patients with total anomalous pulmonary venous return. The procedure is almost certainly equally applicable to the total anomalous venous return in which all the veins empty into an anomalous left superior cava; the so-called "snow-man mediastinum" which is seen on the roentgenogram.

DR. F. J. LEWIS, Minneapolis, Minn.—I would like to congratulate Dr. Watkins and Dr. Gross on their presentation of pioneer work in this field. I would like, also, to express my admiration of the ingenious methods which the other discussers have used to close atrial septal defects without looking at them. Finally, I want to advocate an open method for repairing these defects.

Most of us have been trained in a method of eye-hand coordination by which we do most surgery, and I think we would do best to repair atrial septal defects the same way, employing our vision as long as it remains acute.

(Slide) This shows cases we have operated on to repair atrial septal defects during the past two and one-half years. As you can see, the open technique leads to a slightly lower mortality rate (4 deaths in 33 cases) but it also has other advantages. We are able to identify the complex anatomy typical of some of these defects and place the stitches precisely where they should be placed.

These cases are divided into groups according to a classification we have used. It is based on their embryological development as well as their appearance when we see them in the living heart. We will explain the classification in detail in a presentation during the latter part of the week in Philadelphia. The commonest type is the foramen ovale defect which may have associated anomalous pulmonary venous drainage.

High defects are particularly interesting. I think that some of the defects Dr. Watkins showed in his second or third slide are probably high defects, but they cannot be identified with the finger. The crucial point in their identification is the presence of a

separate foramen ovale below the defect which usually remains closed. High defects are a distinct group. The low defect (the persistent ostium primum is one type of low defect) is fortunately a relatively rare defect. It cannot always be distinguished from a foramen ovale defect by the finger, and I say this from personal experience because we feel all these defects with the finger before we take the trouble to look at them. As has been the case with other people doing this work, our worst experience has been with these persistent ostium primum defects because of postoperative complete heart block. When we learned a little more about repairing them in the third case, we employed the lower part of the left atrial wall, which can be pulled back and used to avoid the conduction system, and in that patient complete heart block did not develop until one month postoperatively. This adds another possible complication, for in these more difficult repairs late scarring may cause damage.

DR. EDGAR DAVIS, Washington, D. C.—I rise not to discuss this but to ask a question. It seems to me that the surgeons operating upon these defects agree that the ostium primum type of interatrial septal defect cannot be satisfactorily closed and concede that they would not operate if the diagnosis could be established clinically. I think we would all welcome some discussion of the possibilities of distinguishing preoperatively between the ostium primum and ostium secundum atrial septal defects.

DR. EARLE B. KAY, Cleveland, Ohio.—From the foregoing discussion it can be concluded that atrial septal defects can be adequately and safely closed by either open or closed techniques. We have preferred the closed technique for we feel that there is less inherent risk to the patient. We have operated upon 19 patients by the closed technique with but one death. This patient was the second one upon whom operation was performed. This patient was 41 years of age and had markedly elevated pulmonary artery and right ventricular pressures associated with arterial desaturation (84 per cent). According to our present standards, she was beyond hope of surgical benefit and should not have been operated upon.

I would like to stress that after pulmonary hypertension and arterial desaturation of significant degree occur, operative closure of atrial septal defects is contraindicated.

DR. WATKINS (Closing).—I want to thank all the discussers. I am very much impressed by their results. It is impossible to discuss all the points raised here, but certain facts should be emphasized.

First, the indications for surgical repair of atrial septal defects are apparently varying in different medical centers. The particular patients upon whom we have performed exploratory operations have either had tremendous flows through their defects or have had debilitating symptoms. Second, we surgeons at present are talking loosely about "pulmonary hypertension." Every patient with a large volume of blood flow through an atrial septal defect will have pulmonary hypertension simply because enormous volumes of blood are being forced through a normal pulmonary vasculature. This innocuous condition must be differentiated from true pulmonary vascular disease, in which there is also a high pulmonary artery pressure but a low flow. The only way one can differentiate these two conditions is by a measurement of pulmonary blood flow and pulmonary arteriolar resistance at cardiac catheterization. The physiologists tell us that systemic and shunt-flow measurements are not valid, because one cannot possibly get a true mixed venous blood sample in the presence of an atrial septal defect, so we neglect all flow observations. However, a Fick systemic flow calculation based upon the superior vena caval oxygen content is a useful rough clinical estimate of left ventricular output, and the more accurate pulmonary flow measurement permits calculation of pulmonary arteriolar resistance. Such observations are necessary to differentiate the common benign condition of pulmonary hypertension due to large blood flow from true pulmonary vascular disease.

We have had no experience with the methods of Sondergaard, Bjork, and Crafoord. However, we have had some difficulty with sutures which pass near the aortic root. The clearances between the coronary artery ostia at that point are rather close; and I am going to try to find out how I can avoid them, from people who have had more experience with

the method. Moreover, defects which lie near the point of branching of the conduction system will present conduction tissue on either side of the septum. Sutures passed to the left of the major conduction system near the anterior edge of the tricuspid annulus can quite easily traumatize the major left bundle branches. I would be interested in knowing how one can avoid these structures.

I believe that Dr. Bahnson's procedure is the solution of an entirely different problem. We have had some experience with diversion of complete pulmonary venous transposition by a somewhat different technique. It seems to us that the major problem in any procedure for this condition stems from the fact that the left ventricle and left atrium are very hypoplastic because of the complete diversion of the venous stream. Consequently, a word of caution might be raised here about the train of events following sudden surgical diversion of all pulmonary venous drainage into such a hypoplastic left atrium and left ventricle.

We have not used hypothermia. We believe that methods for external suturing are quicker and easier for the majority of atrial septal defects although it may take a longer time to describe the actual technique of suture placement.

In reply to Dr. Davis' question about the differential diagnosis of the *ostium primum* defect, we know of no way to diagnose such a lesion definitely, although we are suspicious when there are harsh systolic murmurs along either lower sternal border. In a number of our patients who have had such murmurs, we have found either low-lying defects of the endocardial cushion type or ventricular septal defects with tricuspid regurgitation. Several of our patients who have had complete closure of the defect have had persistence of pulmonary systolic murmurs. The most reliable criterion we have had of complete closure is disappearance of apical diastolic murmurs.

LEFT-SIDED BRONCHOTRACHEAL ANASTOMOSIS

VIKING OLOV BJÖRK, M.D.
STOCKHOLM, SWEDEN

IN most cases of bronchial stenosis of tuberculous origin the corresponding lung parenchyma is destroyed. Then both the lung tissue and its stenotic bronchus have to be resected.

In some cases of stenosis in the main bronchus, one lobe may be found without tuberculous changes. In order to save this healthy lung parenchyma behind the diseased bronchus, Gebauer,[3] in 1949, introduced a method for reconstruction of the tuberculous stenotic bronchus with dermal grafts. In spite of the encouraging results obtained by Gebauer and others,[3, 4, 5, 7] I have never had the courage to suture a dermal graft into a bronchus with a tuberculous stenosis. The main objection is the possibility of reactivation of the bronchial tuberculosis resulting in serious complications.

On the right side, it is questionable if a plastic reconstruction of the main bronchus with dermal graft is ever indicated. The resection of diseased parenchyma and bronchus with a bronchotracheal anastomosis is a more sound surgical procedure.[2, 5, 6, 7]

On the left side, on the other hand, the technical difficulties in performing a bronchotracheal anastomosis have made the dermal graft reconstruction justifiable.[5, 7]

My earlier attempts to do a bronchotracheal anastomosis on the left side have resulted in pneumonectomies. In 1954, Gravel[6] described an end-to-end bronchial anastomosis 1 cm. distal to the carina on the left side. Unfortunately, the patient died four months later due to rupture of an aneurysm of the aortic arch. An inflammatory process had caused destruction of the elastic medial layer of the aorta.

The purpose of this paper is to show that with wide mobilization of the aorta after division of the four upper pairs of intercostal arteries, mobilization of the left subclavian artery, esophagus, trachea, and the central part of the right main bronchus, a direct anastomosis of the left lower lobe bronchus to the trachea is a possible surgical procedure. As a resection of the diseased bronchus with direct anastomosis is to be preferred to a plastic reconstruction with dermal graft, hardly any indication for the use of dermal grafts remains in cases of tuberculous bronchial stenosis.

CASE REPORT

A 32-year-old woman had a tuberculous infiltration in the left upper lobe in 1949. There was progress in 1951, and in 1953 a cavity was visible in the

From Sabbatsberg Hospital: Surgeon-in-Chief: C. Crafoord, M.D., Professor.
Received for publication Feb. 21, 1955.

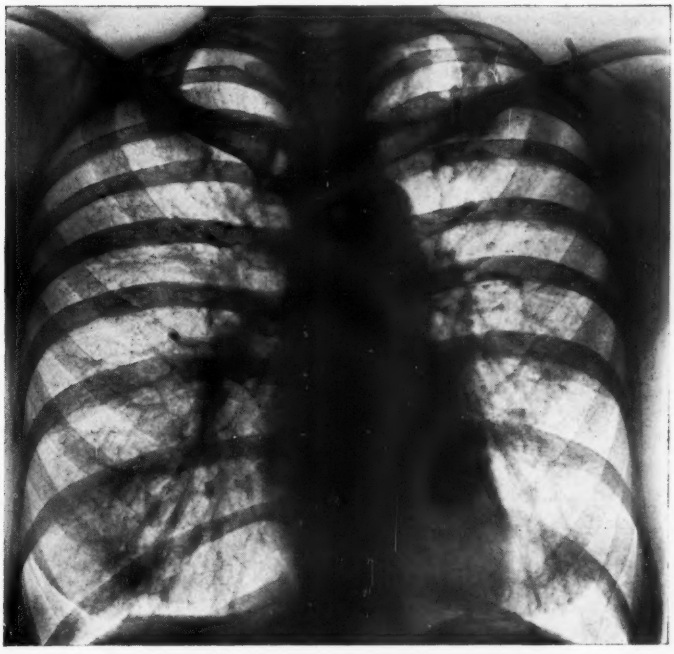


Fig. 1.—X-ray of the lungs demonstrating infiltrations into the left upper lobe.

Fig. 2.—Bronchography before operation which shows a considerable narrowing of the left main bronchus from the carina down to the left upper lobe bronchus. There is no bronchiectasis in the left lower lobe.

left upper lobe. She had a very intense irritating cough. Bronchoscopy performed in January, 1954, showed a circular ulceration in the opening to the left main bronchus which had only a 3 mm. diameter. Later bronchoscopy showed the carina involved in the process. Bronchography showed a considerable narrowing of the left main bronchus from the carina down to the left upper lobe bronchus. The lower lobe bronchus was normal (Figs. 1 and 2).

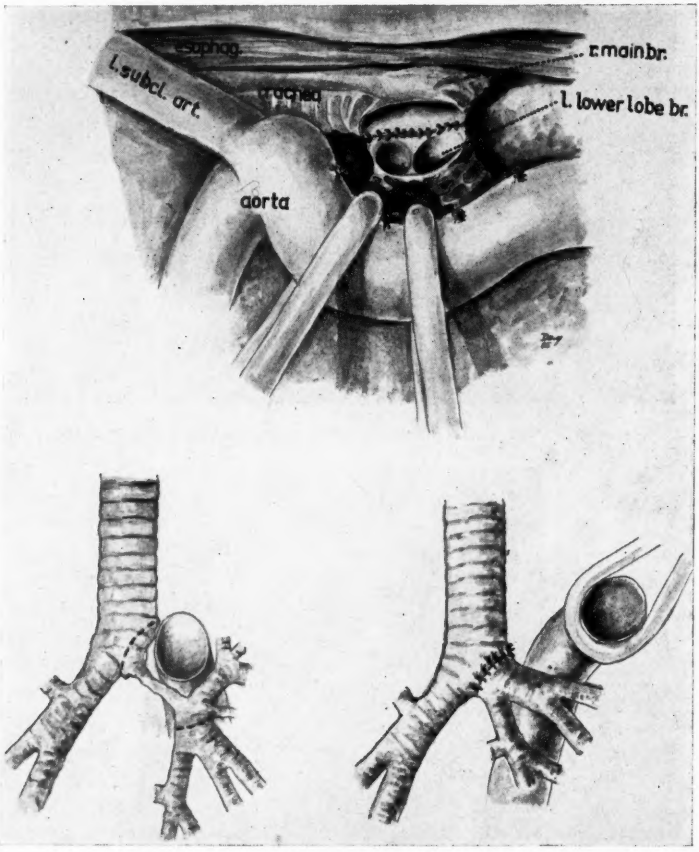


Fig. 3.—Diagram of the operation with mobilization of the aorta and direct anastomosis between left lower lobe and trachea.

At operation (Nov. 17, 1954), the patient was placed in the prone position. A subcostal incision was made under the fifth rib on the left side. Ribs 2 to 6 were divided at the level of the transverse processes. The lung was freed intrapleurally and the upper lobe resected. The pulmonary artery had to be freed from the left main bronchus by sharp dissection from its origin in the main pulmonary artery to its divisions in the basal segmental

arteries. The left main bronchus was dissected as far as possible under the arch of the aorta. Then the four upper pairs of intercostal arteries were divided and the aorta as well as the left subclavian artery was mobilized forward (Fig. 3). The esophagus, the left main bronchus, 5 cm. of the trachea, and the proximal part of the right main bronchus were dissected free.

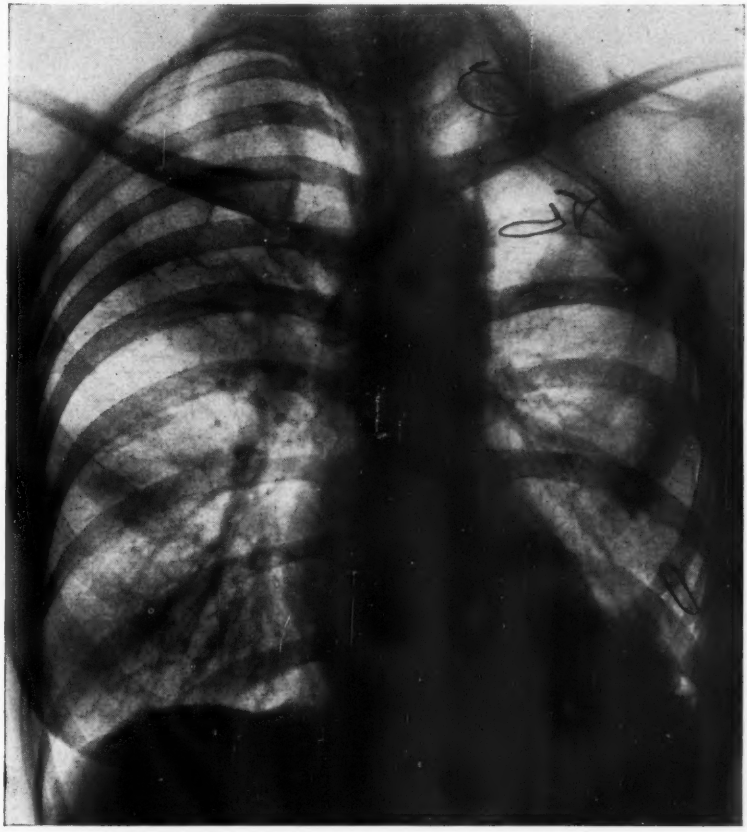


Fig. 4.—X-ray of the lungs 3 months after operation.

The left main bronchus was opened longitudinally in the posterior part. It was impossible to judge, by inspection only, if there was active tuberculosis in the bronchial wall. Now the inferior pulmonary vein was freed but the pulmonary ligament was not divided. The left main bronchus was excised from the trachea and divided just centrally to the bronchus to the superior segment of the lower lobe. The endotracheal tube was pushed down in the right main bronchus and an airtight connection to the right lung was obtained. A Potts' clamp was applied on the lower lobe bronchus which under considerable traction was approximated to the trachea. First a row of iso-

A.

B.

Fig. 5, *A* and *B*.—A bronchography 3 months after operation which shows a patent anastomosis between the left lower lobe and the trachea. The opening to the superior segmental bronchus in the upper angle of the anastomosis to the trachea is also visible. There is no bronchiectasis.

lated over and over No. 00 silk sutures on Atraumatic needles was placed and tied anteriorly. It is better to tie these sutures one at a time, as soon as they are placed. The posterior row of isolated silk sutures was easy to place and tie. The bronchus to the superior segment lower lobe was sutured into the superior corner of the incision in the trachea. The suture line was not covered with a pleural flap. Streptomycin was placed around the suture and the mediastinal pleura closed. The pleura was drained and a small osteoplastic thoracoplasty performed.[1] Bronchoscopy after the operation showed the anastomosis open for the 9 mm. bronchoscope. The patient made an uneventful recovery. The remaining left lower lobe was well aerated during the whole postoperative period and there was no need for a bronchoscopic aspiration.

Three months after the operation the left lower lobe was well aerated (Fig. 4) and bronchography demonstrated a patent bronchotracheal anastomosis. There was no bronchiectasis (Fig. 5, *A* and *B*). Bronchoscopy had to be performed four times for removal of the silk sutures causing granulomas. After removal of the sutures, a well-healed anastomosis was seen seven months after the operation.

Therefore, in a second case of left-sided bronchotracheal anastomosis treated in an identical way and in 3 other cases where the bronchus to the left lower lobe or to the basal segments of left lower lobe was anastomosed to the left-main bronchus, 1 to 2 cm. below the carina without mobilization of the aortic arch, chromic 2-0 and 3-0 catgut was utilized as suture material. Bronchoscopic control of these anastomoses performed with catgut showed a smooth healing without granulomas.

DISCUSSION

As it is possible to perform a direct anastomosis between the left lower lobe bronchus and the trachea, there will not be many cases left for a plastic repair with dermal graft. Excision of the diseased bronchus and end-to-end anastomosis will always be preferred when possible.

The prone position, as advocated by Gebauer for these cases, will facilitate the operation. The most difficult part is the anastomosis. This can be done only after a wide mobilization of the aorta. It is difficult to say how many intercostal arteries can be divided without danger to the spinal cord. We know that in cases of aneurysm of the thoracic aorta all intercostal arteries above the diaphragm have been divided without damage to the spinal cord. It is, however, difficult to estimate the amount of collateral circulation in such a case. Division of the four upper pairs of intercostal arteries will permit a good exposure of the lower part of the trachea, where the anastomosis is performed. This procedure did not cause any harmful effect in the 2 cases reported here. It is preferable to tie each suture after it is placed in the anterior row. The mobilization of the lower lobe is facilitated after division of the pulmonary ligament. The use of chromic catgut for the anastomosis, I

believe, is an important technical detail in order to diminish the formation of suture granulomas. When the anastomosis is completed the suture line should be covered by the mediastinal structures or preferably a pleural flap.

Tracheostomy was not found necessary in these cases.

SUMMARY

Resection of a tuberculous stenosis with a bronchotracheal anastomosis on the left side is preferred to plastic reconstruction with a dermal graft. A wide mobilization of the aorta will make the operation technically possible. The experience of 2 cases with left-sided bronchotracheal anastomoses are reported.

REFERENCES

1. Björk, V. O.: Thoracoplasty. A New Osteoplastic Technique, J. THORACIC SURG. **28**: 194, 1954.
2. Crafoord, C., Björk, V. O., and Hilty, H.: Zur Bronchialresektion und bronchotrachealen Anastomose bei tuberkulöser Bronchialstenose, Ztschr. Thoraxchirurgie, Band 2, 1: 1, 1954.
3. Gebauer, P. W.: Plastic Reconstruction of Tuberculous Bronchostenosis With Dermal Grafts, J. THORACIC SURG. **19**: 604, 1950.
4. Gebauer, P. W.: Further Experiences With Dermal Grafts for Healed Tuberculous Stenosis of the Bronchi and Trachea, J. THORACIC SURG. **20**: 628, 1950.
5. Gebauer, P. W.: Reconstructive Surgery of the Trachea and Bronchi: Late Results With Dermal Grafts, J. THORACIC SURG. **22**: 568, 1951.
6. Gravel, J. A.: Bronchial Resection and Anastomosis, J. THORACIC SURG. **27**: 244, 1954.
7. Mathey, J., and Maillard, J. N.: La chirurgie de la trachée et des bronches. À propos de 22 cas personnels, Semaine hôp. Paris **7**: 1, 1954.

MODIFICATION OF BROCK'S VALVULOTOME RING

GERÓNIMO N. GUASTAVINO, M.D.*
BUENOS AIRES, ARGENTINA

AMONG the better known valvulotomes, Brock's is considered practical because of its small size and easy manipulation. It may be introduced into the auricular appendage and withdrawn with the loss of a very small quantity of blood. It consists of adding a blade to the index finger. There is the inconvenience of having to withdraw the finger from the auricle to apply the instrument. This entails extra trauma to the heart, and although of no practical inconvenience to an experienced surgeon it places extra strain on the heart.

Our object has been to permit the introduction and withdrawal of Brock's valvulotome, leaving the finger in situ. For this we have replaced the original fixed ring with another ring with a spring hinge (Fig. 1).† This allows the

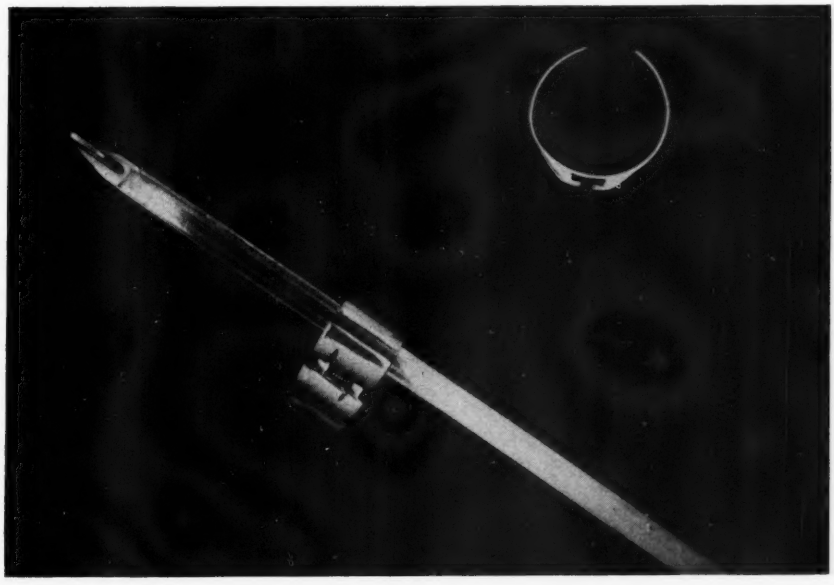


Fig. 1.—With the valvulotome is the newly devised ring; the spring hinge can be seen. The free ring is the Brock's original fixed ring.

Received for publication March 15, 1955.

*Cardiovascular surgeon: Instituto de Perfeccionamiento Médico Quirúrgico, Policlínico Durand, Buenos Aires, Argentina.

†Modification manufactured in the United States by Geo. P. Pilling & Son Co., 3451 Walnut St., Philadelphia, Pa.

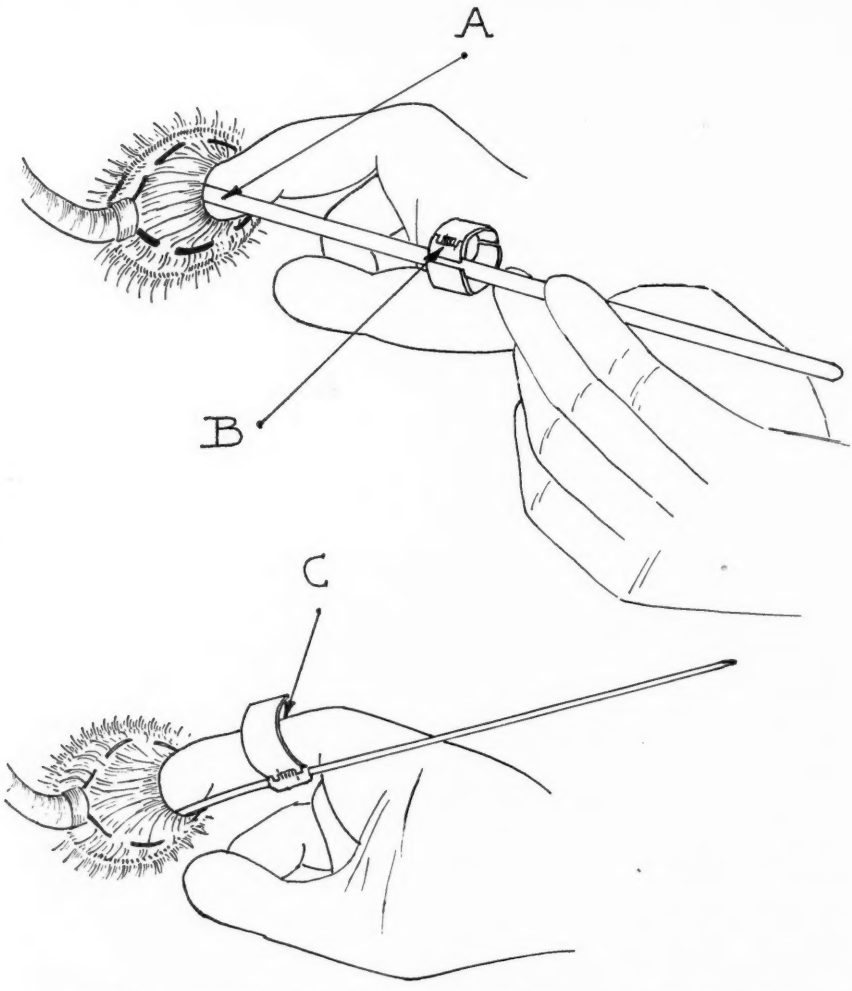


Fig. 2.—*A*, Introduction of valvulotome without withdrawal of the finger from the atrium. *B*, Spring hinge. *C*, Closing of the ring over the finger after the valvulotome has been introduced.

introduction of the valvulotome into the auricle; one then closes the ring over the proximal aspect of the index finger, without having to withdraw it (Fig. 2). It then allows the withdrawal of the instrument once it has completed its object by the opening of the ring that fastens it to the index finger.

We consider this an improvement to this type of valvulotome and on using it we have found it both practical and simple.

Announcements

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The Thirty-sixth Annual Meeting of the American Association for Thoracic Surgery will be held May 7, 8 and 9, 1956, in Miami Beach, Florida. Headquarters will be at the Hotel Fontainebleau.

Requests for Hotel Reservations

These should be mailed directly to Mr. Jack Slone, Director of Sales, Hotel Fontainebleau, Miami Beach, Florida. Please mention this Association, the type of accommodation desired, the date and approximate hour of arrival and departure. If accommodations are desired elsewhere in Miami Beach, please communicate directly with the hotel of your choice.

Thoracic Surgical Forum

The Thoracic Surgical Forum will be continued as a special session at the 1956 meeting. The Forum is primarily for the presentation of short papers on current thoracic research, experimental problems, anatomical studies and modified or new surgical techniques. The inclusion of any considerable amount of clinical material in these reports makes the choice more difficult and the Program Committee reserves the right to reject such papers, or to suggest their transferral to the Regular Program.

Papers at this session will be limited to ten minutes, including the showing of lantern slides or other illustrative material. At the discretion of the President, three minutes may be allowed as the maximum discussion for each paper.

All Thoracic Surgical Forum papers will be published together in one issue of the JOURNAL. Maximum length: 3000 words without illustrations. If illustrated, an appropriate number of words must be subtracted for each cut used.

Abstracts for Papers

Abstracts for the presentation of all papers at the 1956 meeting must be received on or before Dec. 15, 1955, otherwise they will not be considered by the Program Committee.

Abstracts should be labeled "For Thoracic Surgical Forum," or "For Regular Program." The abstracts should contain from 200 to 250 words and accurately reflect the contents of the completed paper.

Five, repeat, five copies of *each* abstract must be sent to the Secretary of the Association, Dr. Paul C. Samson, 3959 Happy Valley Road, Lafayette, California. They should not be sent to the Editor.

Those on the program are reminded that papers presented at the meeting *must* be handed to the secretary immediately after their presentation. If they are not ready at that time there is a chance that they will not be published in the JOURNAL OF THORACIC SURGERY.

Motion Picture Session

Provisions have been made to show a limited number of motion pictures to be selected on a competitive basis. *The films themselves,* together with descriptive material concerning length of presentation, etc., must be received by Dr. Julian Johnson, University Hospital, 3400 Spruce St., Philadelphia 4, Pa., before Dec. 15, 1955.

(Continued on next page.)

It is planned to run the motion pictures in a smaller room at the same time as the scientific sessions. If the films are well labeled, it will not be necessary for the author to be present for comment.

Applications for Membership

Applications for Associate Membership in the Assocation must be received by the Membership Committee not later than Dec. 15, 1955, otherwise, the applications will be deferred for consideration until the 1957 meeting.

Applicants must be sponsored by three Active or Senior Members of the Association. In addition to the signatures on the application form, the sponsors will forward a *separate* letter concerning the applicant directly to:

Dr. O. T. Clagett
Chairman of the Membership Committee
Mayo Clinic
Rochester, Minn.

Sponsors are reminded that *new* letters of recommendation must be forwarded to the Membership Committee should their applicants be held over for reconsideration a second year.

Erratum

In the article, "Traumatic Chylothorax and Thoracic Duct Ligation," by Joseph Goorwitch, which was published in the May, 1955, issue of the JOURNAL, pages 467-479, the following error occurred: In Table III—Cont'd, concerning the case of Seaman, quoted by Goorwitch, it was stated that the treatment was by "thoracenteses followed by ligation of duct in chest." Actually, Dr. Goorwitch should have stated that the treatment was by "thoracenteses followed by plastic repair of the duct without ligation."—EDITOR.